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THE
LARYNGOSCOPE.

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INTRANASAL ENCEPHALOMENINGOCELE. REPORT OF A CASE.*

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The term encephalomeningocele is used because the majority of these tumors contain brain as well as meningeal tissues. Lampert,[1] of Moscow, writing in 1924, refers to two earlier writers, Lissenkoff and Petroff, and says he agrees with them that meningocele as such does not exist. He feels that it is but a secondary manifestation, the primary phenomenon being dystrophy of the brain. The brain tissue tends to disappear and become replaced by connective tissue in these lesions. McGillicudy,[2] in 1942, emphasized this fact and urged the use of the name, encephalomeningocele. Gisselsson,[3] in 1947 expressed the same thought. Examination of the tissue by the pathologist has usually substantiated this fact.

These lesions are usually considered as congenital herniations of the brain and meninges. Guthrie and Dott,[4] however, contend that they are not herniations because the tissue was extruded before the skull was developed and, therefore, was not originally intracranial. These tumors may occur in or near the midline from the occiput to the nasofrontal region. According to Rawling,[5] they may digress from the midline only in the nasofrontal region. Mood[6] says they are usually noted at the sites of the fontanelles, *i.e.*, occipital, frontal,

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sphenoidal or mastoid, and, less frequently, through the cribriform plate of the ethmoid, the foramen cecum, an enlarged foramen magnum or through one of the various suture lines. Ziegler[7] states that more rarely hernias may occur on the sides of the skull or at the base of the skull. According to Lampert[1] lateral hernias are said not to exist at all. In this he agrees with Rawling.[5]

The various theories of pathogenesis have been thoroughly reviewed by Mood[6] and Gisselsson,[3] so they will be referred to only briefly in this report. The deformity begins during embryonic life and is already developed at birth. In 1827, St. Hillaire[15] attributed the growths to adhesions between the dura, brain and skin of the cranium, causing an arrest of the development of the bony vault. Spring,[16] writing in 1854, expressed the belief that increased fluid pressure within the cranium was the primary causative factor. In 1868, Klementowsky[17] stated that diseased bone during embryogenesis caused a malformation of the skull which permitted these deformities to occur. Sternberg,[18] in 1937, points out that the anterior and posterior ends of the neural groove close about a week after the rest of the tube is closed. If these areas remain connected with the skin, the mesoderm from which the skull is formed cannot grow in between. This leaves a dehiscence and a herniation of the brain and meninges. The majority of these herniations are attached to the intracranial contents by a stalk. More rarely the growth of the cranial bone is resumed, closing off the defect imperfectly, and leaving the herniated tissue isolated outside of the cranium.

Encephalomeningoceles are classified according to their location: occipital, sincipital and basal. Von Meyer,[8] in 1890, subdivided the sincipital variety into: *1.* nasofrontal passing between the frontal and nasal bones and presenting in the midline at the root of the nose; *2.* nasoethmoidal passing between the frontal, nasal and ethmoid bones and presenting on the side of the nose; and *3.* naso-orbital passing between the frontal, lacrimal and ethmoid bones and presenting in the medial corner of the eye or in the anterior part of the orbital cavity. Heineke[9] and Safranek[10] classified the basal types as

follows: *1.* sphenopharyngeal passing through the sphenoid down into the epipharynx; *2.* intranasal passing through the lamina cribrosa into the nasal cavity; *3.* spheno-orbital passing through the supraorbital fissure into the orbit behind the eyeball; *4.* sphenomaxillary passing through the supraorbital fissure, then through the infraorbital fissure and into the sphenomaxillary fossa presenting as a tumor on the medial side of the ramus of the mandible or in the zygomatic region.

Whatever their location, these tumors are relatively rare. Gisselsson[3] says one occurs in 3,500 to 4,000 births. Most of these are occipital, 15 to 20 per cent are in the nasofrontal region and 10 per cent are intranasal. Ingraham and Swan[11] collected 546 infants and children with spina bifida or cranium bifidum seen at the Harvard Children's Hospital in 20 years. The ratio of incidence to the number of new cases was 1:4,150. Of these 546 cases, only 84 were the cranial type; 63 occipital, nine parietal, six frontal, five nasal and one nasopharyngeal. Molina and Bertola[12] refer to the same 84 cases. Neither of these reports states whether these cases have been reported elsewhere in detail.

The first case of intranasal encephalomeningocele reported in the literature was that of Richter,[13] in 1813. This was a 60-year-old man with a "polyp" in the nose and a nasofrontal encephalomeningocele. He died following operative removal. In 1890, Von Meyer[8] found five cases in the literature and reported one of his own. He suggested that more cases might well have been reported, but may have been regarded as nasal polyps or other tumors. Fenger[14] reported the first case to be operated successfully in 1895. This was accomplished by opening the nasal cavity, doubly ligating the stalk and packing with iodoform gauze. In 1938, Mood[6] said he had found some 40 cases of congenital herniations of the brain and had seen references to some 30 to 35 cases in the older literature. He stated that "the anterior hernias are usually divided into basal and sincipital varieties, and in the reported cases the two occur with almost equal frequency." In 1942, McGillicuddy[2] reported his case and referred to nine that had been previously recorded. Gisselsson[3] made an exhaustive search of the

literature and reported a case of his own in 1947. He found a total of 34 cases in the literature and gave brief abstracts of 24 of them. The present survey has located and abstracted 12 additional cases and reports one case in detail. There are other cases in the literature in whom the encephalomeningocele was associated with cleft palate and other large cranial defects. These cases are not included.

Clark[19] reported two cases of glioma of the nose in 1905. Although these were apparently true glioma, they are mentioned here because of their marked similarity to encephalomeningoceles. His first case was a two-year-old boy with a rounded tumor the size of a robin's egg at the nasofrontal region. The nose was unusually broad. The left nostril was almost completely obstructed by a pinkish-gray polypoid mass. Biopsy of the intranasal tumor was followed by free bleeding but no cerebrospinal rhinorrhea. The tumor was removed by another surgeon, and the operation was not recorded by Clark. His second case was a 10-week-old boy with a pinkish-gray polypoid mass almost completely obstructing the left nasal vestibule. The site of attachment seemed to be the nasal septum. Three biopsies were taken over a five-month period. Each one caused bleeding, but no cerebrospinal fluid leak developed. No further operative procedures were done. The biopsies on both cases showed gliomatous tissue closely approaching the mucous membrane.

Hopkins,[20] in 1906, described a frontonasal encephalocele with an intranasal portion in an 11-month-old infant. There was a small swelling over the right nasal bone which had been present since birth. Three months previously "polypi" had been removed from the right side of the nose. The right nasal fossa was completely occluded by a neoplasm extending into the vestibule. At operation this extended into the fossa for one-half inch and was apparently attached to the outer wall near the anterior end of the inferior turbinate. He then saw a smooth, grayish, semitranslucent globular tumor at the level of the middle turbinate. Pressure on this was transmitted to the frontal tumor and also from the frontal tumor to the intranasal one. Cerebrospinal rhinorrhea followed. The in-

fant became ill on the fifth day and died of meningitis at the end of two weeks. No operation was done on the frontal tumor or its intranasal extension. Hopkins comments that he finds no reference to an intranasal examination in any of the previously reported cases of nasofrontal encephaloceles and suggests that many of them might have had intranasal extensions. He refers to cases by Chabrely[21] and Demme,[22] in both of which there were similar findings. Chabrely's case had other gross cranial deformities.

Dahmann and Müller,[23] in 1925, report a two-year-old child with a nasal polyp. This was removed by a snare. Meningitis and death resulted in four weeks. At autopsy a dehiscence was found in the cribriform plate with the stalk of a meningocele passing through it.

Guthrie and Dott,[4] in 1927, describe two cases: one was an intracranial glioma with an intranasal extension; the second developed left-sided nasal obstruction from polyps at the age of 41, nine years after a head injury which had caused paralysis of the right arm and leg. Polyps were removed in 1920, 1921 and 1925. The last one was the size of a pigeon's egg, seemingly attached to the middle turbinate. It burst on removal, and about one drachm of straw-colored fluid escaped. The tissue showed nasal mucosa, hyperplastic meninges, and glial cells in three distinct layers. The pedicle of the growth was small, but the exact site of origin could not be determined. No cerebrospinal rhinorrhea or meningitis developed. Two years later no complications had developed, and the nose was in good condition. This encephalomeningocele would seem to be traumatic, rather than of congenital origin.

O'Brien,[24] in 1931, reports a meningocele within the middle turbinate. His patient was a 44-year-old man who had a deviated septum, bilateral nasal polyps and purulent discharge. X-rays showed definite involvement of both ethmoids and sphenoids. In May, 1929, he performed a submucous resection and bilateral polypectomies. In June, he started to do an ethmoidectomy, but after removal of the anterior tip of a markedly enlarged and hard left middle turbinate he en-

countered an immediate flow of bloody fluid. Inspection revcaled a glistening grayish-white membrane the size of the head of a safety match. There was a small perforation in the center from which clear cerebrospinal fluid was escaping. He stated that "examination with a probe showed the glistening membrane to be dura extending well back into the middle turbinate." The nose was packed, but the rhinorrhea tended to persist. This was followed by meningitis from which he recovered. There was no examination of the removed turbinate by a pathologist. Careful analysis of this case raises the question whether this was not actually an exposure of dura rather than the uncovering of a meningocele.

Kaser's[25] (1944) case was an infant with an encephalocele in the right side of the nose showing in the naris. This was seen to move with breathing. The infant was wide between the eyes and showed a bulge on the right side of the nose. X-rays were negative. The growth was biopsied and this was followed by cerebrospinal rhinorrhea. The pathologist reported the tissue as encephalocele.

In 1947, Anderson[26] covered the entire subject thoroughly and presented some very exceptional illustrations of his case. This was an 11-year-old boy who had had a "nasal polyp" removed at the age of four. This was followed by a watery rhinorrhea and meningitis from which he recovered. The rhinorrhea had persisted. During the five months before his referral for operation he had three attacks of meningitis. Examination revealed a spherical, glistening, white mass in the right nasal sulcus almost obliterating the right middle turbinate and apparently originating in the superior meatus. X-rays were normal. A right frontal craniotomy was done, revealing a stalk attached to the right frontal lobe and leading down through a defect 2 mm. in diameter in the cribriform plate. The stalk was cauterized and divided. The aperture was lightly cauterized and closed with a muscle strip and four dural flaps, the whole being sealed over with a strip of gelfoam. The intranasal portion of the tumor was not disturbed and gradually atrophied over the ensuing months. On the fifth postoperative day he had a series of generalized

convulsions from which he recovered. There was no postoperative cerebrospinal rhinorrhea. According to the classifications of Heineke and Safranek this is a true intranasal encephalomeningocele and not one of the sphenopharyngeal variety.

Adson and Uihlein[27] (1949) mention 18 cases of spontaneous cerebrospinal rhinorrhea in their article on intracranial repair of defects in the ethmoid and frontal sinuses. They attribute all of these to nasal meningoceles. X-rays revealed defects in the cribriform plate in three of the 18 cases. They found single fistulous openings in 10 cases and two or more such openings in eight cases. Nasal polyps were observed by their associates in 11 cases. Nine of their cases had had previous operation on nasal polyps. Only two cases are identified, being outlined by Dr. Hallberg. Only these two cases are counted in the present survey. One was a seven-year-old girl with a meningocele in the right side of the nose and the other a man in his forties with one in the left side of the nose. Both of these meningoceles had a whitish look and made their appearance between the septum and middle turbinate. Intracranial repair of the defect in the bone is advocated. Fourteen of the 18 cases were cured. Three cases were failures and two were reoperated without success.

Williamson and Barelli[28] (1951) reported two cases operated by a frontal craniotomy. In the first case the stalk was transected intracranially and the defect in the bone closed by a muscle transplant and gelfoam. The intranasal tumor was not disturbed and gradually reduced in size. The second case was of traumatic origin. The intranasal tumor mass delivered into the cranial cavity at operation and the defect in the cribriform plate was closed by a graft of split frozen rib and dural flaps. X-rays in both cases failed to reveal the dehiscence in the bone. The first case was a five-year-old boy from whom a nasal "polyp" had been removed at the age of nine months. This was followed by a persistent and continuous watery rhinorrhea. The "polyp" had again been removed five weeks before the patient was referred. Following this, the cerebrospinal rhinorrhea had become more profuse.

"Amazingly enough, the child had never had meningitis." The bridge of the nose was very broad and there was a slight internal strabismus. There was a large, gray, glistening mass in the right nasal cavity. Their second case was a four-year-old girl who had sustaind a linear skull fracture into the right ethmoid region four years previously. Temporary rhinorrhea of a few days' duration occurred. Six weeks previously she had had a pneumococcic meningitis which responded to antibiotics. There was a grayish mass high in the right nasal cavity and fluid could be seen to drain from it.

Beyer, Blair and Lipscomb[29] (1951) report a meningocele in a 23-month-old boy. He had developed constant nasal discharge and obstruction of the left nostril at the age of nine months. He had had a nasal operation seven and three months previously and a tonsillectomy and adenoidectomy four months before. The left nostril was occluded by a pale, soft tumor which was continuous with the septal mucosa and was free of any lateral attachment. On packing the nose gently with an adrenalin pack under general anesthesia the tumor disappeared after five minutes. It could then be seen refilling in the olfactory groove. X-rays showed a dehiscence in the cribriform plate on lateral projection. A left transfrontal craniotomy was performed and a dehiscence 1 cm. x 3 mm. was found in the cribriform plate. This was closed by a free pericranial graft. The result was excellent.

Walker, Moore and Simpson[30] (1952) report the case of a baby girl who was observed to have a growth in the right nostril at birth. A portion of this was removed with a snare at the age of 12 hours. Microscopic sections showed it to be an encephalocele. Clear fluid drained from the nose after this biopsy. There was a reddish, soft mass protruding from the right nostril. The mass filled the right nasal cavity and could be seen in the pharynx extending down from the nasopharynx. A tracheotomy was done at the age of one month and a week later a right frontal craniotomy was done. The pedicle was coagulated and divided intracranially and the bony defect closed with a dural flap held in place by gelfoam. Three

months later the mass in the nose and nasopharynx was removed surgically. There was no further cerebrospinal fluid leak and no meningitis developed.

The diagnosis of the intranasal variety of encephalomeningocele is not easily made. There are certain definite findings, however, which should differentiate this lesion from the ordinary nasal polyp and which should put one on guard against doing a casual intranasal operation. If the condition is not properly diagnosed and the growth is removed by a snare there is a prompt leak of cerebrospinal fluid, and this is usually rapidly followed by meningitis. Since this is a congenital deformity, it should be encountered in infancy and early childhood. In Gisselsson's[3] 24 abstracted cases about half were over the age of 10. His own case was 15; however, the symptoms usually dated back to childhood. In the additional cases abstracted in this series, three were over the age of 10 and eight were under this age. These patients have a very widened nasal root with an abnormally wide distance between the eyes (see Fig. 1). This has been noted and commented upon repeatedly since attention was called to it by Fenger,[14] in 1895. The profile (see Fig. 2) may be entirely normal. The growth hangs down from the roof of the nasal cavity like a polyp, but the stalk goes up between the middle turbinate and septum. The stalk of a polyp usually goes up lateral to the middle turbinate. If the growth is large and occludes the nasal cavity, as in the case reported later, it is attached to the septum (see Fig. 3) and a probe will pass up to the roof of the nose only laterally and posteriorly. The probe will pass only part way up along the septum and anteriorly. This point was stressed by Hallerman,[31] in 1932, when he made a plea for a positive diagnosis without biopsy or diagnostic puncture. He felt that either of these procedures resulted in a fatal meningitis. In the formation of this deformity the herniation occurs through the cribriform plate. As the herniation increases in size and descends into the nasal cavity it pushes the nasal mucosa ahead of it. This tends to strip the mucosa from the septum and the under side of the nasal bone. The stalk actually goes up to the cribriform

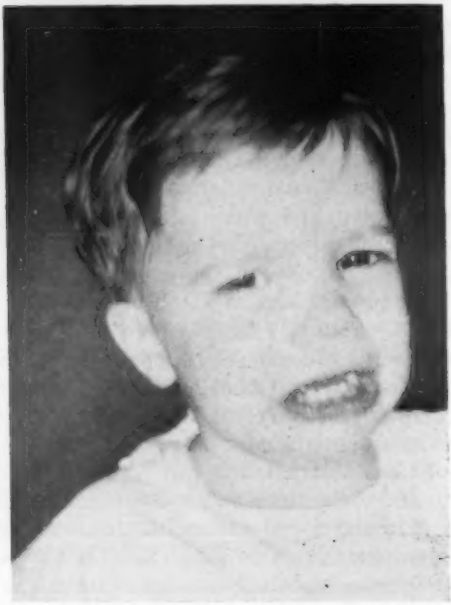

Fig. 1. Note width between eyes.

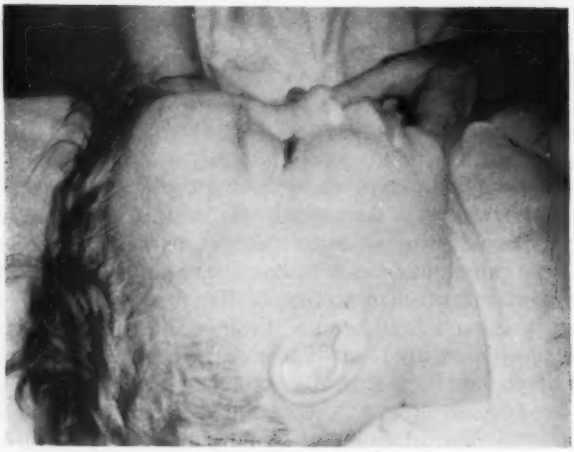

Fig. 2. Normal profile.

plate, but the reflection of the mucosa makes it appear to be attached to the septum. Brain pulsations or change in size with straining or crying has not been seen in any of the intranasal tumors. Both of these findings may occur in the larger occipital and sincipital herniations. Crying during the ex-

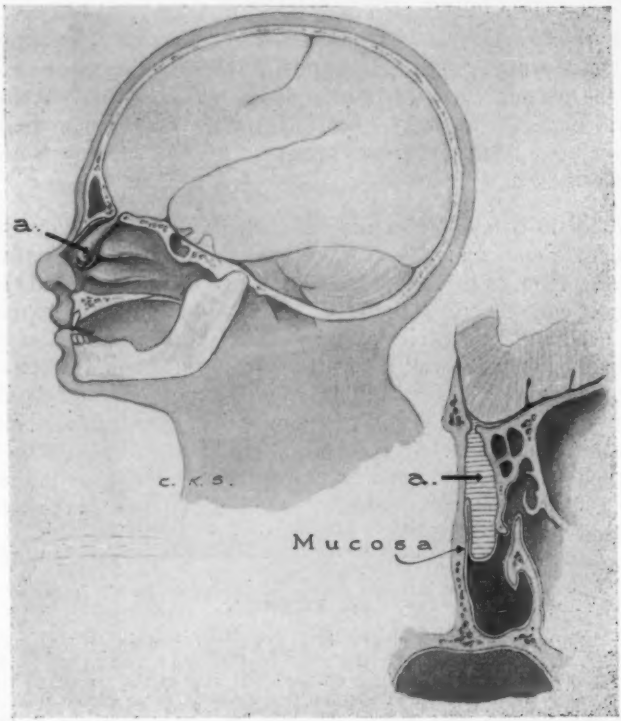

Fig. 3. Encephalomeningocele (a) in nasal cavity. Note reflection of mucosa from septum onto herniated mass.

amination may make the tumor rather firm on palpation and it may not appear cystic except under general anesthesia. Adson and Uihlein[27] list their 18 cases under "spontaneous cerebrospinal rhinorrhea (nasal meningoceles)," stating that nine of them had had previous surgical removal of nasal polyps.

According to Gisselsson[3] cerebrospinal rhinorrhea does not occur unless there has been some trauma, surgical or otherwise. In his series of 25 cases, cerebrospinal rhinorrhea did not occur spontaneously. It was reported only as following biopsy or surgical removal. The same is true of the present series of specifically reported cases.

X-rays rarely show a cranial defect. Six of Gisselsson's[3] 25 cases were X-rayed, and none showed a dehiscence. Four of the present series were said to show defects in the cribriform plate in Roentgenograms. Natanson[32] suggested that a low placed lamina cribrosa might be associated with a meningocele.

Prior to the advent of the sulfas and antibiotics, diagnostic puncture was fraught with grave danger of meningitis and death. Even now it should not be undertaken lightly and then only if one recognizes the possibilities and is prepared to meet all eventualities. Strict asepsis should be observed, and the patient should be well saturated with one of the antibiotics. Aspiration yields a crystal clear fluid, sometimes slightly yellow, with a chemical analysis similar to cerebrospinal fluid. The amount of protein in the cerebrospinal fluid is 15 to 45 mg. per 100 cc. In transudates it is usually less than 2.5 mg. per 100 cc. The amount of glucose in both of these is the same as that found in the blood plasma.[33]

CASE REPORT.

A white male infant, 20 months of age, was referred on April 29, 1950, because of a growth in the right nasal cavity. Four months previously the parents had noticed a bump on the right side of the nose, and that there was a right-sided nasal obstruction. There were no ear complaints. The child did not have many colds, and he was in excellent general health. The ears and throat were normal. The child was very broad across the bridge of the nose and there was an abnormal width between the eyes (see Fig. 1). The right nostril was occluded by a rounded, reddish mass (see Fig. 4). The child cried vigorously during the examination and the mass felt firm to palpation and was noncompressible. It did not increase in size with the crying. Roentgenograms showed a rounded, soft tissue mass in the right nasal cavity with a bending and displacement of the right lateral wall and of the septum. No dehiscence in the cranial vault was visualized. The diagnosis of encephalomeningocele was considered, but because of the apparent firmness of the tumor it was felt that this was a solid tumor like a fibroma or possibly a teratoma.

He was hospitalized on May 21, 1950. The following day he was taken to surgery. Examination under general anesthesia revealed the tumor to be definitely cystic. Probing showed it to be attached by a broad base to the upper portion of the septum and the anterior wall of the nasal cavity. The probe passed up to the vault behind the tumor. The nose and exposed portion of the tumor was thoroughly cleansed and sterilized. Under strict asepsis a needle was inserted into the cystic mass and about

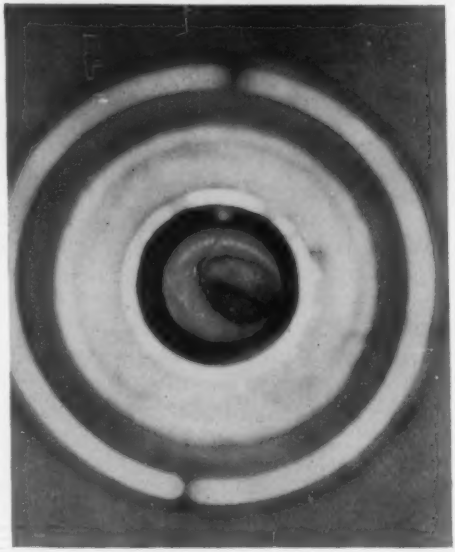

Fig. 4. Tip of encephalomenincocele showing in right nostril.

2 cc. of a crystal-clear fluid was aspirated into a syringe. There was very little bleeding and no subsequent leakage. The removal of the fluid did not appreciably reduce the size of the mass. The patient was returned to his room and he was placed on 300,000 units procaine penicillin a day. Analysis of the fluid showed a total protein of 41 mg. per 100 cc. Urinalysis was normal. Bleeding time was one minute, 15 seconds, and clotting time was two and one-half minutes.

It was felt that the diagnosis of intranasal encephalomeningocele was established. Neurosurgical consultation was obtained, and it was decided to try to prove a definite connection between the nasal cyst and the intracranial contents.

On May 26, 1950, the child was returned to surgery and given a general anesthetic. Dr. William A. Nosik performed a lumbar puncture. The initial pressure ranged from 230 to 240 mm. of water because of forced respirations. When this had become stabilized, pressure was exerted on

the intranasal cyst. There was no change in the spinal fluid pressure. Five cc. of clear, colorless spinal fluid were removed and 2 cc. of indigo carmine were injected into the spinal canal. After waiting 10 minutes, 1.3 cc. of clear, colorless fluid were aspirated from the cyst. There was no evidence of the dye. An equivalent amount of pantopaque was injected into the cyst. After about 15 minutes a series of X-rays was taken with the head held right side up, upside down, and tilted to either side. The pictures showed a large cyst in the nasal cavity filled with pantopaque (see Fig. 5), but no evidence of the radiopaque material inside the cranial cavity. Analysis of the spinal fluid showed 16 mg. per 100 cc. of total protein, 75 mg. of sugar and one W.B.C. Analysis of the cystic fluid showed. 39 mg. of total protein, 75 mg. of sugar, 19 W.B.C. and many

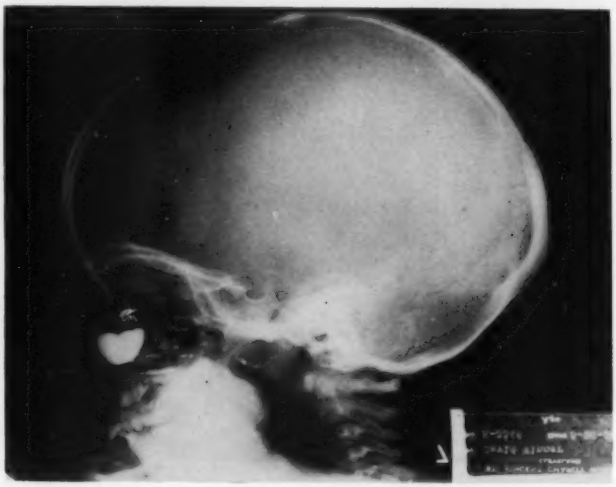

Fig. 5. Pantopaque in encephalomeningocele.

R.B.C. X-rays were repeated on May 23, 26 and 27. None of these revealed any intracranial pantopaque. Three hundred thousand units of procaine penicillin were given intramuscularly daily from May 22 to June 5. The temperature ranged from 36.5° to 37.5° C. except for spikes of 38.4° on May 23, 38.5° on May 29, and 38.6° on June 2. There was a slight cerebrospinal rhinorrhea. He was discharged home on June 5 for a rest before having his operation.

His stay at home was uneventful. He was readmitted to the hospital on June 28, 1950. Blood studies showed 3,720,000 R.B.C., Hgb. 9.5, and W.B.C. 13,100. On June 29 he was placed under intratracheal general anesthesia. The cyst was removed intranasally by the author. The nasal mucosa was incised, exposing the cyst wall, and was then dissected from the cyst in an attempt to remove the cyst in one piece; however, when the dissection reached the upper part of the nasal cavity the cyst ruptured and the loose mucosa and cyst had to be removed with a nasal

snare. The upper portion of the cyst contained white, rather soft tissue. After removal a dehiscence could be seen in the right cribriform plate. The right middle turbinate was atrophic. The nose was packed with gauze.

Dr. William A. Nosik[34] performed a right frontal craniotomy. The dura was dissected from the floor and right olfactory groove. This revealed a dehiscence in the cribriform plate, 1 cm. x 4 mm. in size, through which the intranasal packing could be seen. A small square of tantalum gauze, folded double, to the size of half a postage stamp was placed over the opening. A pad of gelfoam was sealed over this and a larger single piece of tantalum screen was placed over the whole area. The dura was allowed to fall back into position and the wound was closed.

The child tolerated the procedure well. The temperature rose to 39.6° C. on June 29, but was down to 38.9° the following morning and below 38° by evening. He received 400,000 units of procaine penicillin intramuscularly each day until July 6. There was no postoperative cerebrospinal rhinorrhea and he was discharged from the hospital on July 8, 1950. He has been seen several times since then and has been in excellent health. There was some crusting in the right nasal cavity for about a month, but after that the nose has been in good condition.

MICROSCOPIC EXAMINATION.

"The sections on this slide represent a mixture of tissues. Some of the pieces are partially covered by a mucous membrane composed of nonkeratinized stratified squamous epithelium which is continuous with ciliated pseudostratified columnar epithelium. Mucous glands are present in the tissue underlying the latter epithelium. The deep surface of some of these pieces is occupied by a dense collagenous tissue similar to that which will be described later. The latter are composed of a mixture of unmistakable glial tissue and collagenous tissue. The glial tissue shows evidence of compression and shows a focal xanthomatous reaction. The collagenous tissue is arranged in thick, broad, wavy bands. In some locations a loose, highly vascularized, fibrillar tissue is present between the glial and collagenous tissues. In the fibrillar and glial tissues there are widely dispersed calcospherites and there is a foreign body reaction characterized by the presence of multinuclear giant cells. Occasional rounded or prismatic refractile foreign bodies of unidentified nature are situated adjacent to, or within, the giant cells. Considered together, the mixture of tissues is consistent with a meningoencephalocele of the nasal cavity."

DISCUSSION.

A careful analysis of the treatment and results in Gisselsson's[3] series and those given in this report reveals some very interesting and pertinent facts. In the former series, 15 died as a result of meningitis following biopsy or intranasal operation. Of the 10 who survived, two were not operated and only two had an intracranial repair of the bony defect. In the present series only two died of meningitis following intranasal procedures. The outcome of one case that was biopsied is not recorded. Ten cases survived; two following intranasal operation (one of these may not have been an encephalomeningocele) and eight following intracranial closure of the bony defect.

Rawling,[5] writing in 1904, said that a perusal of the reported cases reveals a high mortality. If they survive, the late results are even more unsatisfactory. If not treated surgically they die at an early age. This same thought is expressed by Lampert[1] as late as 1924. He adds that the prognosis is exceedingly bad, whether the patient is treated or not. Both of these authors are referring to the whole group of encephalomeningoceles — occipital, sincipital and basal. In 1906, Hopkins[20] said, "The rate of mortality from operation upon cerebral hernia is high, though less so than before the days of asepsis, and a recurrence often follows, even when the primary operation is successful. If the protrusion is into the nasal cavities the idea of operation is not to be entertained."

These conclusions were made before the days of the antibiotics and were the result of the types of operations which were done on these cases at that time. The high mortality was due to: *1.* not recognizing the true diagnosis of the condition, and *2.* the intranasal biopsy or removal without an attempt to close the bony defect intracranially. The desirability of this latter procedure can hardly be denied. About half of the cases subjected to intranasal procedures alone have died. Of those who did survive, many have had persistent cerebrospinal rhinorrhea with the specter of meningitis hovering over them. The reported cases in which the bony

defect was repaired intracranially have all survived and have had no cerebrospinal leak. In some of the more recent reports the stalk of the tumor has been ligated or cauterized and severed intracranially and the defect closed off. No intranasal operation was done. They report that the intranasal mass gradually atrophied and reduced in size. It would seem likely that this would occur in those masses that were largely cystic. In the case reported by Walker, Moore and Simpson[30] the tumor was very large and more solid. This did not regress in size and was successfully removed intranasally three months later. Because of the possible danger of a delayed removal of the intranasal mass resulting in injury to the tissue sealing off the bony defect, it is suggested that the two procedures be combined and performed at the same time.

SUMMARY AND CONCLUSION.

1. The literature on intranasal encephalomeningoceles has been reviewed, and one case reported.

2. The diagnosis should be established before any operative procedure or biopsy is done. This can be accomplished by observing the following points: *a*. Abnormal width between the eyes. *b*. Cystic polypoid mass in nose with attachment between the middle turbinate and septum or to the septum and anterior walls of the nasal cavity. *c*. Present from birth.

3. Roentgen evidence of bony cranial defect is seldom seen.

4. Brain pulsations and increase in size on crying are not seen.

5. Intracranial repair of the bony dehiscence combined with intranasal removal of the herniated mass is advocated.

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PARACUSIS WILLISII.*

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INTRODUCTION.

Paracusis Willisii is an associated symptom observed clinically in people suffering from bilateral conductive deafness. The paracusic or subject observing the effect of Paracusis Willisii has the sensation that it is possible to hear more easily in the midst of a noisy surrounding than in a relatively quiet ambient. For example, the paracusic reports that the voices of people situated three or four feet away on a moving train can be heard, while it is only with difficulty that the voice of an adjacent person is heard when the train is stopped. Of the many reported incidents, the following is an exceptionally good example to establish the apparent phenomena of paracusis: One paracusic has observed that in a moving automobile a person with normal hearing required a greater intensity setting of the automobile radio than did the paracusic, and the converse when the car was stopped.

The scientific proof of the existence of paracusis by measurement or otherwise reported to date has been practically fruitless. With the aid of modern electroacoustical devices, we have been able to prove such a condition does exist, is of clinical significance and is a measureable quantity.

HISTORY OF PARACUSIS.

The following brief history starts in 1680 when the initial observation of the phenoma was recorded by Willis. Not until

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1875, Buck and later Politzer (1880) proposed the theory that loud extraneous noise shook the ossicular chain, thereby lessening its rigidity, allowing the paracusic to hear better.

In 1877, von Tröltsch voiced the opinion that the increase in hearing was only apparent. Paracusis, he said, was found only in conductive deafness, in which the hearing loss is limited chiefly to the lower frequency range. Since most noise is confined to this region, the paracusic, therefore, is not bothered by the surrounding noise and is able to hear with care the raised voice of the speaker. This same theory was later revoiced by Kranz and Pholman in 1924.

Meanwhile, Urbantschitsch, in 1913, stated that the ambient noise on reaching the cochlea actually increased the sensitivity of the organ of Corti and thereby the acuity of hearing.

Various experiments have been carried out in the past in an endeavor to prove or disprove the existence of paracusis. Some of the more significant are recalled as follows:

Experiment No. 1 — Hastings and Scarff[1] (1928).

Method: An electric audiometer and an electrically driven tuning fork were arranged in such a manner that the sounds of the tuning fork could be transmitted to either ear. The frequency of the audiometer was 728 double vibrations or cycles per second, while the fork had a frequency of 72 cycles per second. The latter was used as a source of extraneous or masking noise relative to that of the former or investigating tone.

Results: a. In two cases of nerve deafness there was found a greater loss of hearing, that is, an increased threshold, for the investigating tone when the masking tone was applied to the same ear, but no change of threshold when the masking tone was applied to the opposite ear.

b. In 60 cases of middle ear deafness it was shown that the threshold was increased whether the masking tone was applied to the same ear as the investigating tone or to the opposite ear.

c. In two cases of otosclerosis the threshold was found to decrease in the presence of the masking tone, that is, a greater acuity of hearing for the investigating tone and thereby a case of true paracusis.

Experiment No. 2 — Shambaugh[2] (1927).

Method: A Western Electric type 2A audiometer was fitted with special ear pieces. The rim of one ear piece was solid and covered with soft rubber to exclude extraneous noise while the rim of the corresponding ear piece was perforated to admit the outside noise. An audiometric investigation of both normals and otosclerotics was carried out on a train both while stopped and while moving.

Results: *a.* The acuity of the hearing of the normal ear was decreased in the presence of the extraneous noise.

b. The acuity of the hearing of the otosclerotic ear was decreased in the presence of the extraneous noise.

Conclusion: The existence of paracusis as a true phenomena was disqualified, as it was by von Tröltsch, and that the occurrence was contributed only to the fact that a normal person naturally raised the intensity of his voice when speaking in a noisy ambient.

Experiment No. 3 — Guttmann and Hamm[3] (1930).

Method: Two electric oscillators with their accompanying amplifiers and attenuators were used to excite a telephone receiver. One oscillator produced a masking or interfering tone of 300 cycles per second, while the other was a variable primary tone of 300, 510, 810 and 2,500 cycles per second. The procedure of the test consisted of determining for both normal and deafened ears the threshold for the primary tone both in the presence and the absence of the masking tone. The intensity of masking used was 50 decibels above the threshold level for the same.

Results: *a.* For both the perceptive and the conductive deafened ear, it was reported that in the presence of the masking tone a slight increase in the intensity of the primary tone produced a greater subjective sensation of hearing than in the normal ear, with the greatest change in the otosclerotic ear.

b. In a few cases of extreme deafness, where only a 20 decibel masking tone was used relative to threshold for the same, there was the occasional ear in which the primary tone was more easily heard with the interfering noise than without the noise.

Experiment No. 4 — McFarlane[4] (1931).

Method: An electric phonograph was used as a source of speech, while a conventional audiometer was used as a source of controlled pure tone. The pure tone was (512 or 1,024 cycles per second) held constant, the speech was applied to the opposite ear and varied in intensity to determine the speech threshold. The reverse of this test was used, in which case the speech served as the masking source.

Results: *a.* The acuity of the normal ear was decreased in the presence of extraneous noise.

b. The acuity of the abnormal ear did increase slightly when small noises were presented.

Conclusion: True paracusis did exist.

EXPERIMENTAL PROCEDURE.

The procedure followed by our investigators, at the Otological and Audiometric Laboratory of the University of Toronto, was to seat the candidate facing the sound source, with head fixed at a distance of 60 inches, as measured from the ear proper to the diaphragm of the sound source (see Fig. 1). The relative position of the subject's head, the sound source,

and the walls of the room was retained for all of the cases investigated. Each candidate was subjected to a precision audiometric examination, including unilateral investigation.

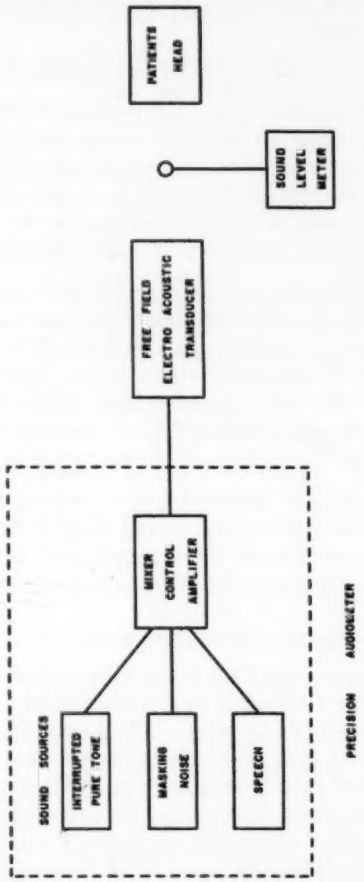

Figure 1

by air and bone conduction and bilateral open field investigation of the threshold response for the pure tones 125, 250, 500, 1,000, 2,000, 4,000 and 8,000 cycles per second, inter-

rupted at a rate of two cycles per second, presenting equal periods of stimulation and silence. The interrupted tones were used for two reasons:

1. To prevent the formation of standing waves in the soundproof room.

2. To produce a second degree of stimulation in the ear.

The bilateral pure tone open field portion of the examination was repeated when an ambient "white noise," the sound level of which in the region of the candidate's head, as measured with a General Radio Type 759-B Sound Level Meter, "c" scale weighted, was 90 decibels relative to 10^{-16} watts per square centimeter, at a frequency of 1,000 cycles per second.

To establish a reference standard for all such tests, 12 candidates, as determined audiometrically, with normal or better than normal bilateral hearing were subjected to this latter procedure. In all, 50 abnormal subjects are reported on, including persons with both bilateral and unilateral hearing defects, classified as early and advanced conductive deafness, mixed deafness and perceptive deafness.

Paracusis was initially believed to be confined to the conductive deafened ear; therefore, sound stimuli between 125 and 2,000 cycles per second were alone used for the pilot investigation of the initial eight persons tested.

RESULTS AND OBSERVATIONS.

The complete observations made for all candidates reported on are on file in the Otological and Audiometric Laboratory. Due to the magnitude of the volume of all such results of the subjects' audiometric examinations, it is only possible in this paper to present the results classified as to the type of deafness determined clinically and audiometrically of the candidates' hearing ability, expressed in decibels relative to the average normal for bilateral open field investigation, with interrupted pure tones in and out of a known noise field.

The results of the initial examination of eight cases of advanced bilateral conductive deafness are as recorded in

Table 1. From these results, Case 1 is presented graphically as shown in Fig. 2, including the interrupted pure tone audiograms by air conduction and bone conduction and the bilateral open field threshold response, expressed relative to the average normal threshold for the test tones, in both an ambient

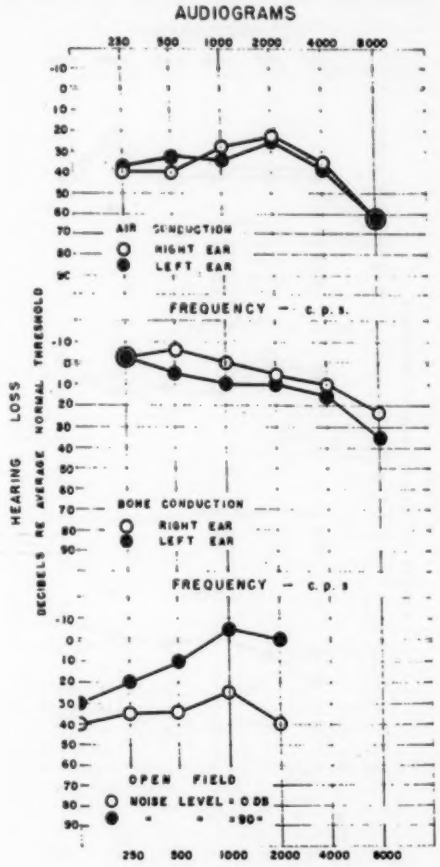

Figure 2 — Case 1

noise field of zero and 90 decibels. The improvement of hearing in the noise field is indicated by the differential in decibels. We see from the lower graph that the least benefit is attained at 125 cycles per second and a maximum benefit is had at the 2,000 cycle frequency.

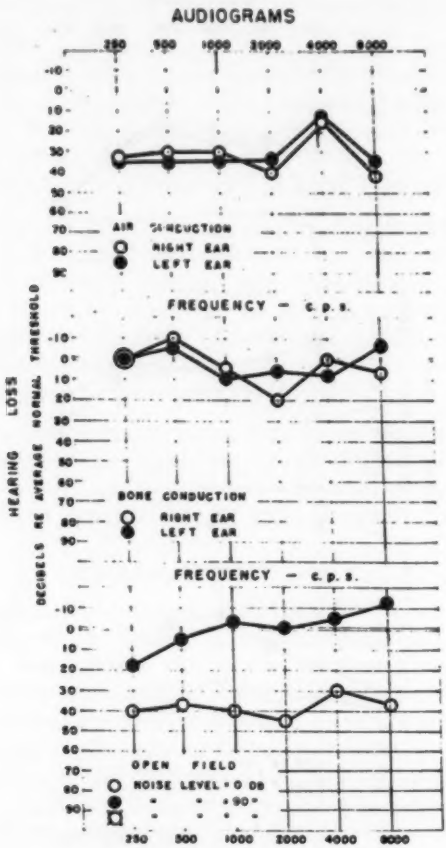

Figure 3 — Case 9

The results of nine cases of advanced bilateral conductive deafness are recorded in Table 2. The graphical presentation of the complete audiometric examination of Case 9 is shown (see Fig. 3). The improvement in hearing as evidenced between an ambient noise level of zero and one of 90 decibel loudness, is as seen in our initial investigation, to indicate

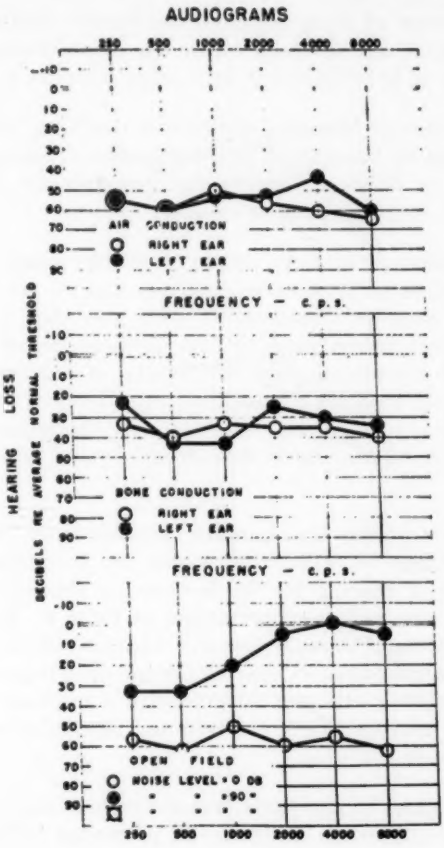

Figure 4 – Case 25

a minimum improvement of hearing for the lower pitch frequencies with a steady increase in improvement to the mid frequencies between 1,000 and 2,000 cycles per second, at which point a normal degree of acuity is had, advancing to a better than normal degree of hearing for the higher order frequencies.

Three cases of early bilateral conductive deafness are as recorded in Table 3, from which the same tendency to improvement of hearing acuity in a noisy ambient is evident.

Four cases of bilateral conductive deafness, with an increased loss of hearing at the 2,000 cycle frequency, are as presented in Table 4. Again the improvement in hearing acuity in a noise ambient is evident.

Four cases of bilateral mixed deafness, made up of an advanced state of conductive deafness and an accompanying degree of cochlear degeneration were recorded in Table 5. The graphical presentation of Case 25 is as shown in Fig. 4, from which we see the same differential of hearing improvement between that for an ambient noise level of zero to that of 90 decibels of loudness, with the only limitation that this improvement of hearing is determined by the basic cochlear sensitivity.

The report of the investigation of seven cases of bilateral mixed deafness in which there was an accompanying increased loss of hearing for the frequencies above 2,000 cycles per second, are included as recorded in Table 6. The graphical presentation of Case 29 (see Fig. 5), as would be expected. shows an improvement in hearing for the pure tones up to and including 2,000 cycles per second, with a marked deficiency for hearing acuity, both in the zero and 90 decibel loud ambient noise fields.

Only two cases of early bilateral nerve deafness were available for investigation. These are as presented in Table 7, in which we see there is no significant change in hearing acuity in or out of these noise fields.

Of the two cases of bilateral nerve deafness recorded in Table 8, the graphical presentation of Case 38 (see Fig. 6) indicates that there is no significant change, or increase in hearing between the lowest frequency and the highest frequency investigated, when the test procedure is carried out

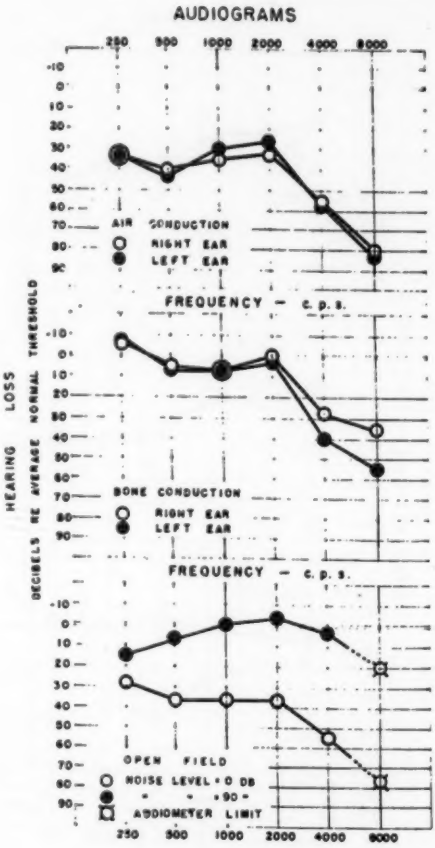

Figure 5 - Case 29

in the 90 versus the zero decibel loud ambient noise field. The interpretation of this graph will be more readily understood from the explanation presented of this phenomena.

The remaining Cases 40 to 50, inclusive, are examples of bilateral hearing defects of which one ear falls within normal sociably acceptable limits, of which there are:

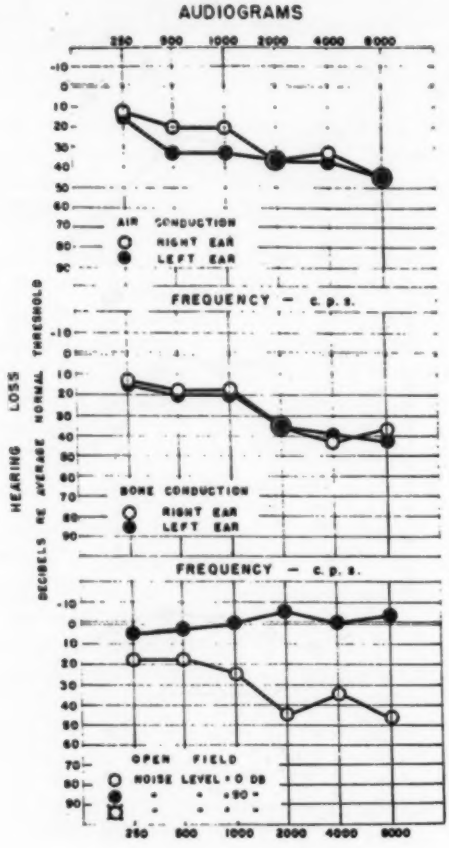

Figure 6 – Case 38

Four cases of unilateral deafness as shown in Table 9.

Five cases of unilateral conductive deafness as presented in Table 10.

Two cases of unilateral mixed deafness as recorded in Table 11.

TABLE-I

INITIAL EIGHT CASES OF ADVANCED BILATERAL CONDUCTIVE DEAFNESS

CASE NUMBER	BILATERAL PURE TONE THRESHOLD – TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES – CYCLES PER SECOND											
	125		250		500		1000		2000			
	MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS			
	0	90	0	90	0	90	0	90	0	90		
1	40	30	35	20	35	10	25	-5	40	0		
2	45	30	45	20	40	7	33	-2	33	0		
3	35	25	40	23	45	15	30	0	35	0		
4	37	25	25	18	28	7	20	-2	28	0		
5	37	28	35	20	30	12	20	-7	30	5		
6	75	50	35	33	40	17	25	5	35	10		
7	27	38	30	23	30	6	35	-2	28	-4		
8	40	30	35	33	40	17	25	5	35	10		

TABLE-2

NINE CASES OF ADVANCED BILATERAL CONDUCTIVE DEAFNESS

CASE NUMBER	BILATERAL PURE TONE THRESHOLD - TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES — CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
9	40	18	38	5	40	-2	45	0	30	-5	37	-12
10	38	15	40	-3	25	-10	38	-7	20	0	27	-17
11	52	18	50	7	30	0	35	-2	38	3	30	10
12	38	18	43	12	38	0	38	-5	25	5	27	-2
13	40	13	23	2	38	-10	33	-7	30	-5	40	0
14	50	25	43	5	35	0	43	0	33	-7	52	0
15	45	28	43	12	48	8	45	5	50	5	52	0
16	43	15	50	10	38	5	35	0	40	-5	27	-10
17	28	20	33	7	28	0	40	-2	20	0	35	0

TABLE – 3

THREE CASES OF EARLY BILATERAL CONDUCTIVE DEAFNESS

CASE NUMBER	BILATERAL PURE TONE THRESHOLD – TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES – CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
18	30	13	33	5	23	-2	40	-5	25	-2	35	-2
19	30	10	33	5	33	3	38	3	18	5	12	-10
20	48	5	38	5	33	0	40	0	38	0	20	-10

TABLE – 4

FOUR CASES OF BILATERAL CONDUCTIVE DEAFNESS WITH INCREASED LOSS AT 2000 c.p.s.

CASE NUMBER	BILATERAL PURE TONE THRESHOLD – TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES – CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
21	47	22	57	12	43	0	70	10	53	8	57	3
22	40	15	48	2	33	-7	45	3	28	3	13	-10
23	25	10	25	0	25	5	48	-10	33	-7	15	-15
24	48	18	50	10	53	5	63	3	43	3	22	-7

TABLE – 5

FOUR CASES OF BILATERAL MIXED DEAFNESS TYPE NO. I

CASE NUMBER	BILATERAL PURE TONE THRESHOLD – TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES – CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
25	58	33	62	30	60	20	60	5	55	0	62	-5
26	≥75	≥50	≥80	≥42	73	2	78	17	80	20	≥77	≥20
27	45	13	35	0	35	8	40	0	40	0	47	0
28	40	20	43	10	40	3	50	3	38	3	45	-5

TABLE-6

SEVEN CASES OF BILATERAL MIXED DEAFNESS TYPE NO. 2

CASE NUMBER	BILATERAL PURE TONE THRESHOLD – TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES – CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS		MASKING LOUDNESS-DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
29	28	15	37	7	38	0	38	-2	65	3	75	20
30	50	30	50	12	53	10	83	20	93	20	77	20
31	43	18	45	7	48	5	78	0	85	5	77	20
32	38	13	43	5	25	3	48	0	63	3	62	20
33	43	18	38	10	30	8	33	10	48	5	52	20
34	48	25	50	10	38	-5	45	0	50	3	57	-5
35	43	20	45	7	33	-5	48	0	60	-5	69	0

TABLE – 7

TWO CASES OF EARLY BILATERAL NERVE DEAFNESS

CASE NUMBER	BILATERAL PURE TONE THRESHOLD – TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES – CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS		MASKING LOUDNESS – DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
36	-2	-2	-5	-5	-10	-3	3	-3	30	0	47	20
37	-2	0	-10	0	-7	0	10	-2	15	5	32	10

TABLE 8

TWO CASES OF BILATERAL NERVE DEAFNESS

CASE NUMBER	BILATERAL PURE TONE THRESHOLD—TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES — CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
38	18	5	18	2	25	0	45	-5	35	0	47	-2
39	38	30	43	20	38	10	30	7	18	7	35	5

TABLE — 9

FOUR CASES OF UNILATERAL DEAFNESS — ONE EAR PRACTICALLY NORMAL

CASE NUMBER	BILATERAL PURE TONE THRESHOLD — TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD TEST FREQUENCIES — CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
40	25	8	23	3	23	6	25	1	20	6	20	4
41	25	5	28	0	18	3	35	0	35	0	5	-5
42	40	18	30	5	23	5	30	8	17	3	33	20
43	23	2	20	2	18	3	43	3	45	5	77	10

TABLE - 10

FIVE CASES OF UNILATERAL CONDUCTIVE DEAFNESS

CASE NUMBER	BILATERAL PURE TONE THRESHOLD – TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES – CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
44	40	23	38	10	28	3	18	0	28	0	35	-10,
45	35	15	38	5	25	-2	25	-2	20	-7	32	-15
46	43	25	45	10	35	5	53	0	38	8	42	7
47	38	28	30	17	25	5	23	-2	13	-2	22	20
48	40	20	37	5	35	0	45	0	43	0	40	-10

TABLE - II

TWO CASES OF UNILATERAL MIXED DEAFNESS TYPE NO. I

CASE NUMBER	BILATERAL PURE TONE THRESHOLD — TONE INTERRUPTED DECIBELS RE AVERAGE NORMAL THRESHOLD											
	TEST FREQUENCIES — CYCLES PER SECOND											
	250		500		1000		2000		4000		8000	
	MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS		MASKING LOUDNESS - DECIBELS	
	0	90	0	90	0	90	0	90	0	90	0	90
49	33	15	28	0	25	0	38	3	48	5	60	3
50	47	20	43	7	43	3	53	3	60	0	65	20

EXPLANATION OF PARACUSIS.

Since the complete comprehension of this phenomena is impossible at this time, we do, however, postulate the following:

In the presence of any sound, the human being has the following natural reflexes:

1. Intensifying the voice when speaking.
2. Reducing the sensitivity of the ear by
   a. Tensing the tympanic membrane.
   b. Increasing the transmission stiffness factor of the ossicles.
3. The reduction in sensitivity of the ear as a whole to the response of sound at high frequency to that of any loud, low pitched, masking or interfering sound.

With this in view it is possible to state that the sensitivity of the normal ear in the presence of an intense ambient noise will be reduced considerably, from the effects of the tympanic membrane and the ossicles, for frequencies below 1,500 cycles per second and from the masking effect for frequencies inclusively and particularly above 1,500 cycles per second. The threshold obtained for the average normal ear under such conditions of ambient noise is, therefore, the reference plane or standard of hearing for tests in a known ambient noise field.

This standard, as is any other, is absolutely arbitrary, when all the limits of the test are stipulated. The comparison of the normal ear is made first with the ear suffering from pure conduction deafness which may be due to either an impediment of motion of the ossicles and the oval window, or to obstruction of the round window with complete freedom of motion of the ossicles, or to obstruction to both the round and oval windows to various degrees of magnitude, with the resultant natural reduction to the transmission of any sound of low frequency. This may safely be said to be of a greater

degree of absorption than that capably induced by the normal ear. Hence, a greater degree of absorption of sound reaching the cochlea exists, thereby lessening the masking effect of the noise and maintaining a greater degree of sensitivity of the cochlea to the higher test frequencies.

Since the standard of reference in a known ambient noise is the sensitivity of the normal functioning cochlea, therefore, an equality and even greater sensitivity to frequencies, say, greater than 1,500 cycles per second, can be expected of the conductive deafened ear, while a natural reduction relative to the normal threshold will exist for frequencies lesser than 1,500 cycles per second. As was observed in actual measurement, the conductive deafened ear showed a practically linear decibel loss from 0 decibels at 1,000 cycles per second, to 20 decibels at 250 cycles per second and a linear decibel gain to minus 15 decibels at 8,000 cycles per second, as predicted.

From the results obtained, it would appear as though the internal ear responds to intense stimuli in a different fashion than at the threshold intensity. This possibly is associated with the function of the inner rods of Corti, which form part of the tunnel of the organ of Corti.

When a comparison is made with the case of true perceptive deafness, the results that should occur are obvious, namely, if degeneration or complete dysfunction of the sensitized nerve endings has resulted, an apparent loss or lessened response to that of the normal ear under similar ambient stimulus would be present. In general, no effect should show in the response to the lower tones as the tympanum and ossicles are functioning normally, until the degenerative damage extends to involve the low frequency reception of the cochlea. Since the number of cases of true perceptive deafness at the disposal of the laboratory for purposes of testing were limited, the confirmation of this theory is only approached from the results available. The actual measurement necessary in such a procedure under the condition of a loud ambient noise, for any case of perceptive deafness, is difficult due to the pain developed, the recruitment effect and the retention through-

out the entire test of the undivided cooperation of the patient; therefore, the existence of paracusis can only be expected of persons suffering from some form of bilateral conductive deafness and can be stated as being of an apparent condition only, since such a person is basing his comparison in terms of a normal functioning cochlea of some arbitrary reference sensitivity. At low intensities the reference sensitivity of the cochlea of both normal and abnormal are alike, but due to the absorption to sound transmission by the conductive abnormality, an appreciable hearing loss or difference in threshold exists relative to the normal ear as a whole, under similar conditions. At high noise intensities another but a lesser degree of sensitivity of the cochlea prevails for the normal ear to that in the quiet, while in the abnormal ear this lowering change in sensitivity is not so great as that of the normal ear; however, the ears of both have apparently the same degree of practicable overall sensitivity under the condition of an intense ambient noise. As the ambient noise is reduced to zero magnitude, the normal ear appreciates the change in intensity of the ambient noise without realizing the change in overall sensitivity which takes place, since the rate of change of sensitivity is quite small. The abnormal ear, on the other hand, realizes not only the change in intensity, but also the overall change of sensitivity required to render the ambient noise inaudible; therefore, the rate of change of sensitivity is sufficiently great to be appreciated. The converse of this applies equally as well. Such abnormal ears, therefore, do have an apparent increase in sensitivity from the quiet to the noisy ambient, whereas the normal ear does not have any apparent change in sensitivity.

Attention should be drawn to the saturation effect that can be expected to be reached for all types of ears when ambient noise is increased further and further. Prediction of the intensity of the ambient noise which would produce this saturation effect is that intensity of noise which would cause pain in the normal ear. An optimum, therefore, likely exists as far as the paracusic is concerned.

CONCLUSIONS.

The following conclusions may therefore be drawn:

1. Paracusis does exist.

2. Paracusis is to be found only in people suffering from some form of conductive deafness.

3. The conductive deafened ear responds equally as well, if not better than, the normal ear, in the presence of an intense noise field, for tones above 1,500 cycles per second with a slight loss below 1,500 cycles per second.

4. The rate of change of sensitivity of the abnormal conductive deafened ear relative to the normal ear is large, between the respective thresholds for the test sounds in the quiet to the same sounds in an intense ambient noise field.

5. An optimum of pure conductive deafness as observed, for example, clinically in otosclerosis, will afford the greatest possible change, or the extent of the increase in sensitivity observed by the paracusic.

6. An optimum exists relative to the magnitude of the ambient noise field intensity that may be applied to the paracusic before a reduction in sensitivity of the ear occurs.

7. Paracusis is unobserved by the ear afflicted with perceptive deafness, as the reduction in overall sensitivity is not appreciated any more than in the normal ear.

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ADULT TONSILLECTOMIES USING PENTOTHAL SODIUM, CURARE, PONTOCAINE AND NOVOCAINE AS THE ANESTHETIC.*

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A paper on "The Use of Sodium Pentothal Anesthesia in Adult Tonsillectomies" was presented before the Eastern Section of the Triological Society, January, 1949, at Boston, by Irl H. Blaisdell.[1] His report stimulated our interest in this new technique. As a result of certain modifications, the combined opinions of both anesthetists and surgeons, the following procedure has been devised as presented in this paper.

The adoption of a new technique or a modification of an old one is at times an extremely difficult accomplishment; however, any procedure which will reduce the incidence of complications to the patient and make an otherwise major procedure easier and safer for the surgeon should have its merits.

The gravity of tonsillectomies both as to morbidity and mortality is rapidly declining, due to improvement in surgical technique and postoperative care. Flick and associates,[2] reporting in 1929, stated that in 172 cases of pulmonary abscess, 120 patients, or 70 per cent, were postoperative. The abscess in 97 cases, or 57 per cent, of the series appeared after tonsillectomy. In 1938, Morrison[3] stated 20 to 30 per cent of all cases of pulmonary abscess were due to tonsillectomies.

The presence of aspirated blood in tracheal secretions was shown by Hara,[4] also by Steele and Anderson,[5] in 100 per cent and 96.6 per cent, respectively, in their series of tonsillectomies with the patient in the supine position. Contribu-

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ting factors toward this finding could have been inadequate suction during the operation, and unskilled assistance in depressing the tongue. Using the procedure to be outlined, we have had less than 1 per cent of the patients with blood in the tracheal secretions at the termination of surgery.

During the past three plus years 189 adult patients (14 years and older) have had tonsillectomies in which sodium pentothal, curare, pontocaine and novocaine was the anesthetic. It has been surprising, the alacrity with which these patients choose sodium pentothal as the anesthetic when given their choice. The features which please the patient are the lack of fear of fighting the anesthetic, either ether or local novocaine; their desire not to participate in the operation, and their fear of suffering pain during the operation. The additional features of no nausea or vomiting and a quiet postoperative period is appreciated by the patient.

For the surgeon the desirable features are rapidity of induction, for the patient is asleep in less than 30 seconds. The throat is perfectly quiet during the operation; there is no straining; there is less bleeding; there is no limit to the amount of suctioning one may wish to do, for there is no anesthetic being withdrawn.

Features of the technique which have made this operation less of a strain to the surgeon include sitting at the head of the table, the use of the Goode headlight, tilting the operating table from a level to a head low position of 10 degrees, and the additional hyperextension of the head. The use of the Davis-Crowe or the McIvor mouth gag is desirable.

Sodium pentothal was first introduced in 1934. Its use in throat surgery became popularized about 1945 when it was used alone,[6,7] later with nitrous oxide,[8,9] and with cocaine,[10] but always with intubation. Curare was added, as reported by Robinson,[11] who found that curare decreased the reaction time from an average of 75 minutes to 25 minutes and less. Blaisdell[1] stimulated renewed interest in this subject.

In a recent report Dillon[12] states that sodium pentothal is a somnifacient and should be considered as such. It is not an

anesthetic in the true use of the term, for it does not have the additional properties of analgesia. For this reason it is most important to use a local anesthetic, especially when operating in the oral and pharyngeal areas.

By the addition of pontocaine and novocaine as a local anesthetic to the above procedures we have eliminated the use of the endotracheal tube. Where the procedure is carried out meticuously there has been no laryngospasm. In this series of cases laryngospasm has not been present.

Preoperative Medication: This procedure is only used on patients over 14 years of age. Patients 14 to 17 years of age are given atropine sulfate 1/300 gr. (0.2 mg.) and morphine sulfate 1/8 gr. (8.0 mgm.) hypodermically; adults are given morphine sulfate 1/6 gr. (10.0 mgm.) and scopolamine hydrobromide 1/200 gr. (0.3 mgm.) hypodermically 30 to 60 minutes preoperatively. Atropine may be used in place of scopolamine hydrobromide.

When the patient arrives at the operating room and has been transferred to the operating table, his throat is sprayed with pontocaine solution 1 per cent or 2 per cent by the anesthetist, using the Rawbotham atomizer. This increases the time the pontocaine has to take effect, and also when given before the instillation of sodium pentothal lessens the tendency to laryngospasm.

An intravenous normal saline solution is then started. Then a mixture of sodium pentothal 2.5 per cent solution, 9 cc.; plus syncurine, a synthetic curare, 1 cc. is injected in the I.V. tubing; using several cc. to put the patient to sleep, more being added as needed to control the patient. It is unusual to use more than 10 cc. of the mixture per patient.

Within less than 60 seconds the patient is asleep. The patient is draped, the head of the table is lowered so the body is at a 10 degree angle, and the Davis-Crowe mouth gag is inserted. The tonsils area is injected with novocaine solution, 1 per cent, with adrenalin hydrochloride 1-1,000, six minims to the ounce of novocaine solution. Usually less than 10 cc. of novocaine solution is used for both sides.

There are several modifications which I have adopted, that are not essential, but which, I found, are desirable. I check the adenoid area before starting the tonsillectomy, using the Hasslinger soft palate retractor and a laryngeal mirror. An excellent view of the Eustachian tubes and any possible adenoid growth is thus obtained.

Following the tonsillectomy and the control of bleeding, the curved suction tip is inserted into the nasopharynx and the blood there is suctioned out. There is usually a large sized clot in this region.

An airway is placed in the patient's mouth and he is transferred back to his room. He is awake within 20 minutes without the nausea and discomfort of an ether anesthetic. An ice collar is placed on the patient's neck, and the care given local tonsillectomies is then carried out.

In this procedure an endotracheal tube is *not* used. The danger normally encountered in the use of sodium pentothal, which demands the use of an endotracheal tube, has been eliminated. The larynx has been desensitized by a pontocaine spray preoperatively; there is no foreign body, as blood or mucus, encroaching on the vocal cords, throwing them into laryngeal spasm. Blood lying in the nasopharynx, the dependent part of the pharynx, does not get as high as the arytenoids. The larynx is in view during the greater part of the operative procedure.

CONCLUSIONS.

1. A modification of the usual routine for tonsillectomy is presented.
2. The procedure consists of the use of the Davis-Crowe mouth gag; the head low position; sodium pentothal, curare, pontocaine and noocaine as anesthesia.
3. An endotracheal tube is not used.
4. Using this technique on 189 adult tonsillectomies has presented no undesirable reactions.

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CORRECTION.

In the June, 1952, issue of THE LARYNGOSCOPE, page 577, "An Osler Item," by Harris P. Mosher, M.D., line 12, paragraph 1, should read: "but that was all, because Dr. Osler and Mrs. Mosher, who went as far as London . . ."

THE FUTURE OF OTOLARYNGOLOGY.*

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Four years ago in his Presidential Address before this Society Dr. Lyman Richards initiated some sober thinking about the future of our specialty. His thesis, "Otolaryngology in Transition,"[1] recognized the changes wrought by the advent of chemotherapy and the antibiotics, and the inroads that other fields of practice seem to be making into our specialty.

Since then, others[2-4] have analyzed our status. Their pronouncements in writing have indicated both optimistic and pessimistic outlooks. A few weeks ago, the general medical public had a stimulus to think about otolaryngology as a specialty because of an editorial in the *Journal of the American Medical Association*, entitled "The Future of Otolaryngology."[5]

Some among us have expressed concern over the doubts this editorial will place in the minds of prospective recruits for our specialty; others seem to believe it will add to our desire for further self-appraisal which will ultimately have a beneficial result. This latter group will go along with the thought expressed in the closing sentences of this editorial to the effect, "That only in a climate of continuously free and open discussion can a valid, objective assessment of the future of otolaryngology be made. Otolaryngologists have much to gain by threshing out the problem calmly and deliberately rather than by ignoring it." Dr. Nash apparently had this in mind when he planned this part of the program months ago.

In September, 1951, the annual report by the American Medical Association on "Approved Interneships and Residen-

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cies in the United States"[6] indicated that one out of three of the approved residencies for otolaryngological training was unfilled. Today, there is an apparent shortage of trained otolaryngologists to fill spots in group practice, or for association with an older otolaryngologist who needs help, or for one who wants to discontinue a combined practice of ophthalmology and otolaryngology and relax into the less rigorous demands of ophthalmology alone. There is a tendency on the part of those older men practicing the combined specialty to advise young men against choosing otolaryngology singly as a specialty. We also hear from the combined specialists in rural areas and from small clinic groups the lament that combination eye, ear, nose and throat specialists are not being trained any more.

In discussing the present status of otolaryngology as a specialty, I wish to think with you about the following:

1. Has the actual number now in preparation for our specialty changed substantially in the past decade?
2. The trend toward specialization has increased. Has otolaryngology shared in the increased numbers entering specialties?
3. Is there need for continued specialization in ophthalmology and otolaryngology for small communities?
4. Why have other fields of practice been making inroads into our specialty?
5. What is our future?

Actual figures are available to establish the facts concerning the first two questions. The following figures (see Table 1) show the number of programs approved by the American Medical Association in 1940[7] for residency training in otolaryngology, in ophthalmology, and in the combined specialty, and in addition the total number of residencies offered:

Eleven years later the approved combined residency is no longer listed. (There will apparently be some approved training programs of two years each in ophthalmology and oto-

laryngology.) In 1951, the number of approved programs and residencies offered in either otolaryngology or ophthalmology were as follows:

TABLE 1.

1940	Approved Programs	Residencies Offered
Otolaryngology	63	173
Ophthalmology	51	131
O. and O.	40	100

If we count the approved programs in 1940 that were combined as the equivalent in number of approved residencies in otolaryngology, these figures indicate that in 1951 there were 47 per cent more approved programs and 49 per cent more residencies offered in otolaryngology than there were in 1940. In September of 1951, it was reported that 33 per cent vacancies existed. If 33 per cent of the 408 approved residencies were vacant in 1951 that means that 273 were filled, the exact number of approved residencies available in 1940. We do not know how many of the 273 residencies available in 1940 may have been vacant. Probably some of them were. These figures do indicate however, that the actual number in preparation in otolaryngology when these figures were obtained in 1951 is as large as it was a decade ago.

It has been often stated that the trend toward specialization has increased. Just how great this trend has been is probably not realized until we examine the figures. The latest figures on approved residencies, issued by the American Medical Association, are shown in the following table and compared with the figures for 1941:[8]

The report stated that in September of 1951 there were 25 per cent over all vacancies in these residencies.

Some of the vacancy percentages that exceeded otolaryngology are: neurology, 39 per cent; pathology, 39 per cent; physical medicine, 58 per cent, in addition to the more limited

lines of specialization, such as in cardiovascular diseases, 47 per cent; in malignant diseases, 50 per cent, and in pulmonary diseases, 45 per cent.

Some additional statistics that are of interest for comparison are those related to interneships and the number of medical school graduates.

A 32 per cent vacancy rate was reported in approved interneships in 1951.

In Table 1 it was indicated that in 1940 there were fewer residencies offered in ophthalmology than there were in otolaryngology and there were fewer approved programs. Table 2 shows that this situation had become reversed in 1951. In addition, there were 21 per cent vacancies in ophthalmology in 1951 as compared to the 33 per cent vacancy rate in otolaryngology.

TABLE 2.

1951	Approved Programs	Residencies Offered
Otolaryngology	152 (47% increase)	408 (49% increase)
Ophthalmology	172 (88% increase)	551 (138% increase)

The trend toward specialization has increased as shown in Table 3, but otolaryngology has not participated, relatively, in the increased numbers entering the specialties.

TABLE 3.

Year	No. of Approved Residency Programs in All Specialties	Total Number of Residencies Offered
1941	616	5,233
1951	4,462	19,364
	(624% increase)	(290% increase)

TABLE 4.

Year	No. of Hospitals Approved for Interne Training	No. of Interneships	Medical School Graduates
1941	735	8,182	5,275
1951	821	10,044	6,135
	(10% increase)	(22% increase)	(16% increase)

The military service demands probably have something to do with this, as it does with all other residency vacancies. Table 5 shows that there were over 10,000 more physicians in military service in 1951 than in 1940.

TABLE 5.

Year	Physicians in Military Service
1940[9]	2,386
1945[10]	60,000
1951[11]	13,000

Most of us feel certain, however, that some are not considering otolaryngology as a specialty because of its alleged doubtful future.

Is there a need for combined specialization in ophthalmology and otolaryngology for small communities?

It has been my observation that the physician who specializes in both ophthalmology and otolaryngology in small communities, and in many instances in larger centers, is really a sort of general practitioner in these specialties. As far as I can determine, the combination of these two fields was largely an American creation. Prior to the sulfonamide and antibiotic era, this combination specialist in the smaller centers was engaged in fitting glasses, treating external eye diseases, opening eardrums, doing mastoid surgery for acute disease rather poorly, and administering a great deal of treatment for nasal disorders. His chief rhinologic surgery was an occasional submucous resection. He managed the anterior dislocation of the septum poorly. In the laryngological area his chief surgical activity was on tonsils and adenoids. Complications occurring in the disorders he treated were usually sent to a better equipped specialist in a larger center. Problems of hearing loss, chronic suppuration of the middle ear and mastoid, problems of vertigo and tinnitus, the more formidable of the external ear disorders, the persistent complaints of the patient with the self-diagnosis of sinus disease, the difficult appearing surgical septum problem, external nasal deformity, tumors, disorders in the oral cavity, disor-

ders with chronic pharyngeal and laryngeal symptoms, and in most cases the problems involving endoscopic procedures were usually referred to a better equipped specialist in a larger community. Today the character of his otolaryngological activities has been altered a great deal because of modern chemotherapy and the use of antibiotics.

The combination specialist in the larger community usually exhibited a major interest in ophthalmology or in some phase of otolaryngology and became identified with that interest. A substantial majority of those combination specialists made no attempt to cover the complete fields of ophthalmology and otolaryngology and today their otolaryngological activities consist mostly of tonsil and adenoid surgery, an occasional submucous resection and the office treatment of the common ear, nose and throat complaints. Many spend a majority of their time fitting glasses.

Probably a typical example of this changed situation is represented in the city of Minneapolis. Twenty years ago there were two men limiting their work to ophthalmology and two to otolaryngology. Thirty-nine practiced the combined specialty. Today in this city of over half a million population with another half million adjacent to it, there are exactly 31 specialists limiting their work to either ophthalmology or otolaryngology, and 31 who list themselves as "eye, ear, nose and throat specialists." I would estimate that 75 per cent of the work of any one of this combination group is concerned with the eye. With three exceptions, none do any laryngeal or endoscopic work, few are interested in modern temporal bone surgery, the problems of audiology or medical otology, surgical rhinology, tumors, etc.

Those who lament the passing of the "eye, ear, nose and throat specialist" are concerned about the absence of this type of specialist in a community the size of which apparently would not be large enough for an ophthalmologist or an otolaryngologist to practice either specialty singly.

I cannot speak for ophthalmology, but I doubt that the welfare of any community is in jeopardy because this community

lacks the residency of an otolaryngologist. As a rule, the combination specialist is not equipped to handle the really urgent emergencies in the field of otolaryngology, such as respiratory tract foreign body. The modernly trained general practitioner can manage most of the otolaryngologic situations of a minor nature and transportation is so efficient in the present day that major problems have relatively easy access to the otolaryngologist who is well equipped.

Probably a parallel situation exists in ophthalmology.

It is probable, however, that there will continue to be a place in the smaller communities, at least for some time to come, for the physician who wishes to practice in the combined fields. I can see no reason why he should need Board certification any more than special certification is needed for general practice. His training will probably be acquired largely as it has been in the past. It was usually based on an experience in general practice supplemented by intensive short courses in various aspects of the two specialties.

Why have other fields of practice made inroads into our specialty?

The two activities in our specialty that are usually mentioned first when we decry the seeming invasion of our field of work are tonsil and adenoid surgery, and bronchoscopy. Now, tonsil and adenoid surgery has always been a part of general practice and it is in the domain of one who calls himself a general surgeon. If figures were available, I wonder if these would show that it is the general practitioner or surgeon who has actually reduced the number being done by the otolaryngologist. Certainly the public has become more specialist-minded if those figures showing the trend toward specialization in the past decade have any meaning. In some communities it is true that the pediatrician has taken up tonsil and adenoid surgery and this is an inroad. I doubt that this will spread. I know of no teaching institutions associated with medical schools where the pediatric training embraces tonsil and adenoid surgery, and I doubt that it ever will as long as a respected otolaryngologist is on the job. As a rule,

specialists in other fields who are true specialists recognize the fact that the lymphoid tissue of the pharynx should be better managed in relation to the health of the upper respiratory tract by a well trained and experienced otolaryngologist than it can be done by one to whose activities this is just a sideline.

Perhaps there is less tonsil and adenoid surgery to do because the indications have been reduced. The theory of focal infection is not all that it was thought to be 25 years ago. Neither does infection carry the same hazard because of the safety that chemotherapy and the antibiotics have brought.

One thing that does bother me, however, is the fact that there are busy otolaryngologists who welcome the opportunity to rid themselves of the doing of this surgery, even though they wish to continue being known as throat specialists. If tonsil and adenoid surgery is better done by the specially skilled, then why not continue to retain this responsibility?

It is my personal conviction regarding the matter of invasion of the field of endoscopy by the chest surgeon or internist that when this has occurred it has in many instances been a matter of a situation in which the assistance furnished by the otolaryngologist has been inadequate to these other specialties. I want to cite a personal experience in this regard. It offers a solution to some of these problems.

Twenty years ago at the University Hospital in Minneapolis, there was dissatisfaction in the medical and surgical departments in the matter of getting prompt diagnostic procedures performed in the field of bronchoesophagology. The surgical department then sent one of their staff away to learn the technique of pulmonary and esophageal endoscopy. After he came back and was getting in our way, we decided that we were better equipped than he was. We began a campaign of talking, demonstrating, offering to be right on the job when emergency situations arose (and these were quite common in chest surgery before modern anesthesiologists were available) and in the course of a very few years our competition practically vanished.

A similar situation was present when the otolaryngological service at the Minneapolis General Hospital was taken over by our department at the end of World War II. Dr. Robert Priest is in charge there, and by offering a superior endoscopic service we have practically recovered this work which was being done by an internist. Similarly, the endoscopic work at Ancker Hospital in St. Paul, one of our affiliated services, has been captured from a chest surgeon by Dr. Jerome Hilger and his associates, Dr. Hochfilzer and Dr. Goltz.

Now, I do not mean to imply that the otolaryngologist should try to stop the chest surgeon from doing bronchoscopy. The latter, if he is equipped by training and experience to do bronchoscopy, should use all the means that he can personally to make an accurate diagnosis for himself; however, the fact that chest surgeons do their own bronchoscopy is not going to end this activity for otolaryngologists. In the first place, the number of chest surgeons will obviously be limited. It is only in the larger cities that one can exist in this specialty. I have a notion that the chest physician who is not a surgeon will continue to prefer the services of the otolaryngologist who has demonstrated his special skill in technique, a knowledge of chest pathology and bronchographic interpretation and the fact that he carries a stethoscope in his pocket and uses it. I am sure that the chest surgeon in teaching institutions is not going to do bronchoscopy unless the department of otolaryngology falls down on the job.

In some of these discussions about our changing status and the future, it has been claimed that other specialties are also invading our field. The allergist, the maxillofacial surgeon, and even the psychiatrist are mentioned. I disagree with these ideas.

Allergy is a relatively new specialty, but otolaryngologists slept with the idea that all rhinitis was due to infection, while voices like those of Hansel and others at one time fell on rather deaf ears. Today, we know that the otolaryngologist

who develops an interest in the allergic problems of our specialty does a much better job of managing these problems than do the allergists who seek to go alone.

Maxillofacial surgery as a specialty has always been, I believe, extremely limited. Most, if not all, of those who practice it as a specialty exhibit degrees in both dentistry and medicine. Facial injuries, tumors of both jaws and of dental and oral cavity origin, and cleft palate work comprise their field. Some include a certain amount of facial plastic surgery. I can't see where they have necessarily invaded our specialty, but I also can't see why the otolaryngologist who is interested should not equip himself to care skillfully for the work done by the maxillofacial surgeon. No one can be critical of the otolaryngologist who knows what he is doing and does a good job.

The psychiatric angle to this usurpation is even less apparent. Otolaryngologists worked a long time before waking up to the fact that nasal stuffiness, many forms of head pain, certain throat symptoms, etc., are often not the result of primary disease at these sites that I have mentioned. The competent otolaryngologist of today may not be doing as many surgical procedures as he might be doing if he was unaware that psychiatric factors play a rôle in a number of symptoms within our field of activity. In that sense, the former degree of surgical activity has lessened a bit, but it seems only proper not to accuse the psychiatrist of invading our field, but rather to blame ourselves for our shortcomings.

What is our future?

At the 1951 meeting of the Teachers' Section of the Academy, this question was raised, "Have the Opportunities in Otolaryngology Increased or Decreased in the Past 15 Years?"

Dr. Howard House spoke for otology, Dr. Jerome Hilger for rhinology and Dr. Howard McCart for laryngology. Each reviewed the present day activities in his field and concluded that the possibilities are so numerous for the broadly trained

otolaryngologist that, singly, he had difficulty in adequately covering our specialty. These discussions are to be published in the next issue of the *Transactions* of the Academy.

I am sure that most of you will agree with these conclusions. But now comes "the $64 question": where is one to get a training that will equip him adequately in the various activities which we now regard as part of our specialty? Unfortunately, there are not enough institutions where such training can be obtained. Lierle[12] commented on this at the meetings in San Francisco in 1950 when he said:

"The most glaring deficiency in our training seems to be lack of uniformity in the scope or field of otolaryngology in our teaching institutions. The Board of Otolaryngology required that candidates be examined in all phases of otolaryngology, peroral endoscopy, maxillofacial surgery, and surgery of the neck exclusive of the thyroid gland. It has been revealed that only one-half of the institutions teach the entire field of our specialty. In a number, the operative procedures consist merely of tonsillectomies and submucous operations. . . . I think the future of otolaryngology is in the hands of the country's teaching institutions."

This last statement of Lierle's, to my mind, sums it all up in a single sentence. Let us, therefore, take a look at the composite picture and think about the following:

1. The grass roots of our specialty are in the undergraduate students. We urgently need improved teaching programs in many of our universities. The Teachers' Section of the Academy has adopted minimum standards for this. We need missionaries for this work, but these must and should be found through initiative at a local level.

2. Where a high level of undergraduate teaching is found, there also will be found or will develop a good graduate program. The combined appraising facilities of the American Medical Association, the American College of Surgeons and the Board are reviewing the programs for residencies. Approval will be limited to those institutions where an adequate

three-year program of graduate training can actually be offered within that institution or its affiliations. Some protest that a three-year training program is too long. Those who protest would probably be the first to insist that our specialty requires as much specialized knowledge as a well trained surgeon or internist must have. Superior preparation in those specialties requires as much and usually more than can be obtained in a three-year residency.

3. These objectives are not to be gained overnight. At least 10 years will pass and probably a second 10 years before a sound basic program of undergraduate and graduate instruction will be attained in a majority of our institutions. When this has been accomplished, we need have no apprehension about the flow of talent into our residencies or about the regard that other specialties have for us. Again quoting Lierle: "If we attain excellence as specialists, we need not concern ourselves about what others do in our particular field."

I close with a statement made by Gordon Hoople:[13] "Surveys . . . critical reviews, those are all-important, but far outclassing them is the job we ourselves have got to do."

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MALIGNANT DISEASES OF THE PARANASAL SINUSES WITH A CASE REPORT OF PRIMARY CARCINOMA OF THE ETHMOID LABYRINTH COMPLICATED BY NASAL POLYPOSIS.*†

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Malignant diseases of the paranasal sinuses are now not infrequently reported in the otolaryngological literature. This is due to our alertness and improved diagnostic methods. The incidence for malignant tumors of paranasal sinuses varies with reporting authors. According to Watson[1] there was 0.44 per cent of paranasal sinus cancer among patients admitted to the Memorial Hospital in New York within 10 years. Ewing[2] is of the opinion that neoplastic diseases of the nasal passages and accessory sinuses are of frequent occurrence, and that about 2 per cent of all cancer cases are in this area. The same author in the review of a large number of malignant diseases found that 1.84 per cent occurred in the antrum of Highmore. Seelig[3] states that in a series of 2,724 patients with cancer, admitted to the New York City Hospital from 1935 to 1946, there were eight cases, or 0.22 per cent, of carcinoma of the maxillary sinus. In Ringert's[4] series the nasal cavity was involved in 49 per cent of the cases, the ethmoid labyrinth in 43.1 per cent, the alveolar process in 45.4 per cent, the orbit was invaded in 22 per cent, and the antrum of Highmore was involved in all instances.

According to Watson[1] the maxillary sinus is five times more susceptible to cancer than the other sinuses combined. The ethmoid sinus is next most frequently involved, the frontal and sphenoid being less frequently engaged in the order mentioned.

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†From the Yorkton General Hospital.

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The commonest malignancy encountered in this region is a squamous cell carcinoma. Geschickter[5] found that squamous cell carcinoma of the nose and its accessory sinuses rarely develops in patients under 40 and is most frequent in the fifth to seventh decades. The youngest patient in his series was a 15-year-old girl. In Watson's[1] series, a boy of four years. Males are more susceptible than females. Fabricant[6] found that malignant tumors of the paranasal sinuses are seven times more frequent in the male than in the female.

REPORT OF A CASE.

The interest in the case being reported was aroused by the combination of malignancy of the ethmoid sinus with nasal polyposis which masked the serious primary lesion. Unanswered questions remained as to whether a nasal polyp developed into malignancy and whether the ethmoid labyrinth was primarily or secondarily involved as was the orbit.

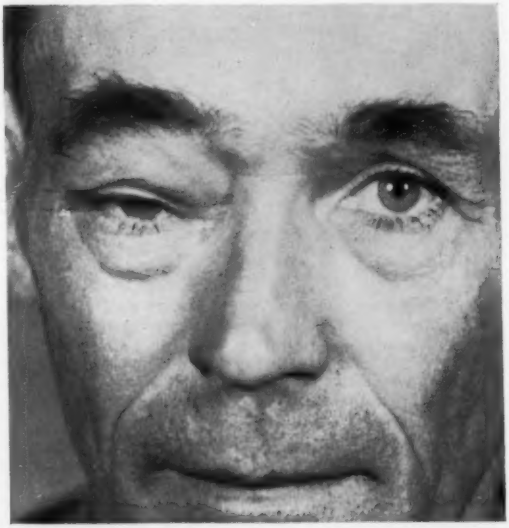
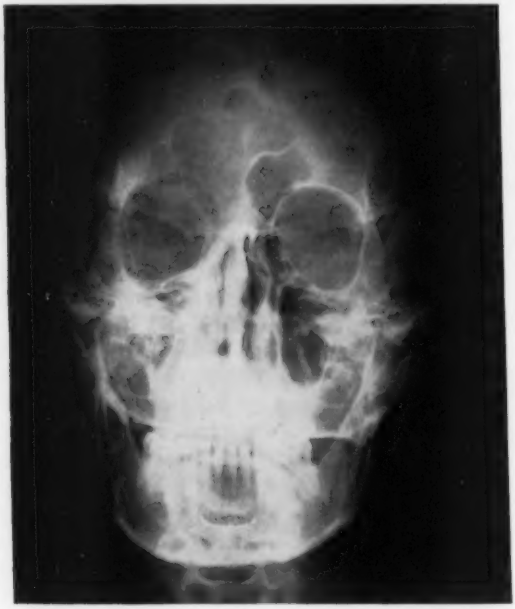


Fig. 1. Preoperative photograph showing the swelling of both right eyelids, reduction of the palpebral fissure, shifting of the right eyeball and bulging of the area of right inner canthus.

The patient was a 37-year-old, well developed and well nourished white man, referred to me in April, 1951. The presenting features were right nasal obstruction and swelling of the right eye. He gave a history of painless, gradually increasing obstruction of the right nostril with some whitish-yellow, never bloody discharge. The growth of the obstruction was slow, developing over a period of two years. This was associated

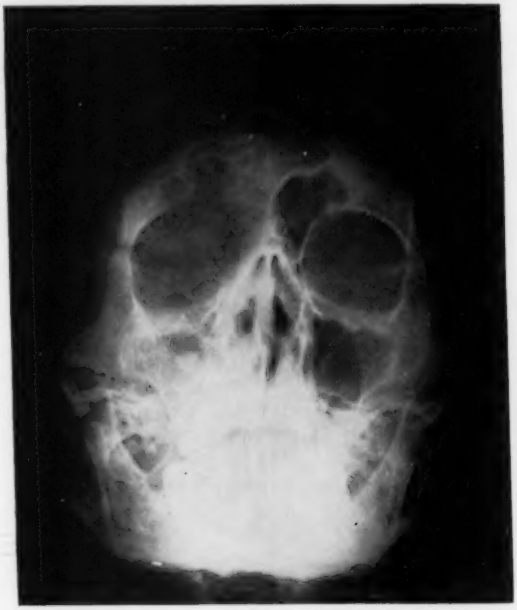


Left.

with mild dull headaches, varied in intensity and frequency, limited mostly to the right frontal region, coming on in spells for one hour and easily relieved with aspirin. In January, 1951, he noticed a definite swelling of the right eye, which was tender on palpation. Four weeks prior to his office call the patient complained of blurred vision, especially while reading. This was accompanied with lacrimation of the right eye. No diplopia was noted.

Appendectomy in 1936 was the only significant item in his past medical history. No childhood diseases were recalled. No history of injury. Family and social history irrelevant. He has lost no weight. On examination the patient was cooperative and his general physical condition was good. The significant findings were confined to the right nasal chamber and right eye. The right nostril was packed with numerous pink-grayish, shiny, not bleeding on touching, nasal polypi which completely obstructed the right nasal airway.

The nasal polypi were protruding between middle and inferior turbinates and covered with mucopurulent discharge. The inferior turbinate was hidden by the polypi. The nasal septum was deflected to the left. The left nasal chamber appeared to be normal with normal olfaction. No offensive smell. No tenderness on palpation over the right maxillary sinus, some tenderness over the right side of the nasal bridge. The nasopharyngoscopic examination was negative in all respects. The hard pal-



Right.

Fig. 2. Two Roentgenograms showing markedly increased density of the right frontal, ethmoid, and maxillar sinuses, and the right orbit. The films also demonstrate the extensive erosion and destruction of the superior nasal quadrant of the right orbital margin, roof of the orbit, and anterior plate of the right frontal sinus.

ate was normal in appearance as well as on palpation. Transillumination of the sinuses disclosed a very dense right maxillary and frontal sinuses, all other sinuses being clear.

Right Eye: Both upper and lower lids were edematous, and the palpebral fissure reduced markedly. No considerable exophthalmos could be visualized, but the eyeball was deviated downward and somewhat outward and depressed. Pain and tenderness on palpation round the inner half of the right orbital margin. In the orbit a firm but resilient mass contiguous with bone was palpable, extending along its internal wall and being most

prominent near the inner canthus and along the roof of the orbit. The pupil was circular and central and equal with the left one. Its reactions were normal. The extraocular movements in all planes were intact. Fundus examination indicated no abnormalities. Vision without correction, 20/100. *Left eye* normal.

The results of the physical examination were otherwise within normal limits, with palpable but not enlarged right-sided lymph glands of the neck. Routine laboratory work irrelevant, serologic tests for syphilis being negative.

Diagnosis: In search for the diagnosis two X-ray films of the accessory paranasal sinuses were taken in Yorkton General Hospital and described by Dr. L. C. Hacking as follows: "There would appear to be marked increased density affecting the very large right frontal sinus, ethmoid area, antrum, and orbital cavity on this side. How much of this may be due to the tumor process and how much due to secondary inflammatory reaction I am unable to determine from the films or, indeed, radiologically from any other method. There would seem to be extensive erosion and destruction of a large portion of the superior nasal quadrant of the right orbital margin and including portion of the roof of the orbit and probably anterior plate of the right frontal sinus. The extent of the area of destruction cannot be definitely determined from these films. . . ."

Fig. 3. Histological section of the polyp covered on one surface by pseudostratified transitional epithelium and on the other surface by epithelium of more squamous type.

For diagnostic purposes and in order to establish drainage of the affected sinuses a right nasal polypectomy was performed. About 15 polypi were removed from the right nasal chamber. The nasal polypi originated from the right middle meatus and middle turbinate. This turbinate appeared to be soft, fragile and rugged. Its anterior pole was partially

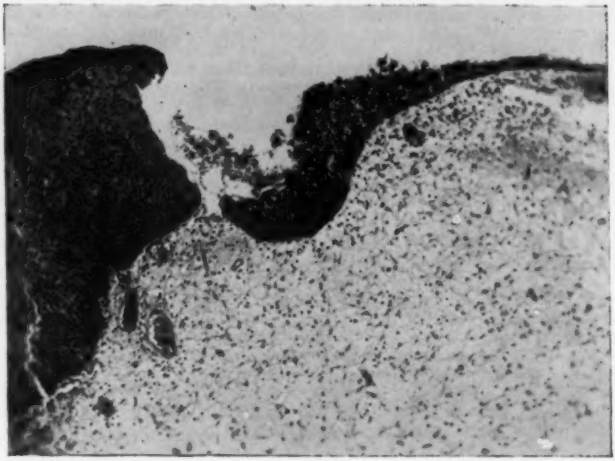


Fig. 4. Microscopic section of higher magnification of the same polyp giving more detail.

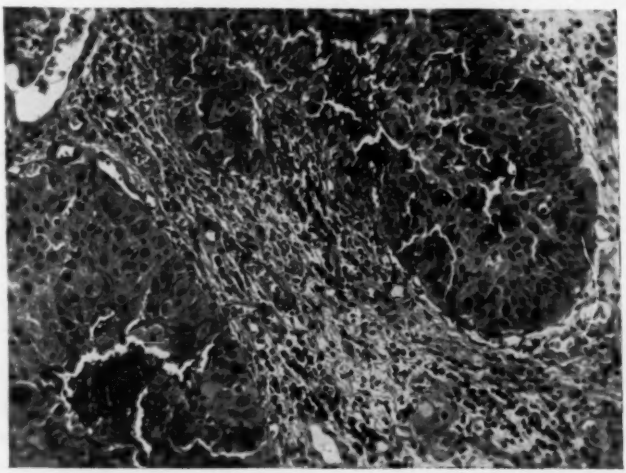


Fig. 5. Section of the roughened surface of the same polyp. The histopathological diagnosis given was epidermoid carcinoma with invasion of the stroma by the malignant epithelial cells.

amputated and the bulla ethmoidalis opened. Thick yellow-greenish purulent discharge was encountered in the ethmoid labyrinth. This operation was accompanied by heavy bleeding which stopped under tamponade. One of the removed nasal polypi had a deep red, hard and rough surface, its other surfaces being grayish-pink, elastic and smooth, as in normal polyps. The pathologist's report (Dr. J. D. Stephen) was as follows: Gross description: "Specimen consists of several pieces of mucoid polypoid tissue. The largest measures 3 cm. in length and 1.5 cm. in diameter and has an irregular warty surface." Microscopic description: "This is a nasal polyp covered on one surface by pseudostratified transitional epithelium and on the other surface by epithelium of a more squamous type. The roughened surface noted on the gross examination proves on histologic examination to be an area of epidermoid carcinoma with invasion of the stroma by the malignant epithelial cells."

As the diagnosis of malignancy was thus established, further differential diagnosis from orbital periostitis and cellulitis, osteomyelitis of the forehead from frontal sinus infection, pyocele, orbital abscess, benign nasal growths, tuberculosis, rhinoscleroma and Boeck's sarcoidosis became unnecessary. Syphilis was excluded by negative serologic tests. Although it was important to locate the primary site of the malignant growth in our patient, this was difficult and uncertain because of the anatomy of the part involved and late stage of the malignant disease.

According to Capps[7] primary malignant neoplasm occurs only in the maxillary and ethmoid sinuses, but the site of origin is usually uncertain because when the malignancy is diagnosed it has already extended far beyond the primary site.

The symptoms of cancer of any paranasal sinus are those of advanced disease and may consist of headache, unilateral nasal obstruction, unilateral nasal discharge, epistaxis, swelling of the cheek and palate, edema of the eyelids, displacement of the eye (proptosis), epiphora and diplopia. The differentiation as to the site of primary lesion is not easy. Schall[8] is of the opinion that the symptoms of the malignant growth in the antrum of Highmore depend upon the location of the tumor. If it originates in the hard palate or the superior alveolar process there is swelling of the palate, loosening and falling out of the teeth and even fistulization into the antrum. If the malignant growth originates from the posterior superior antral wall there is paresthesia or anesthesia of the cheek of the affected side. None of these specific antral symptoms were noted in our patient, and as the puncture of the antrum, performed after polypectomy,was negative we may assume that the maxillary sinus was not the primary site of the malignancy. The haziness of the same sinus seen in the X-ray

films may be attributed to an inflammatory reaction. More detail could be obtained by cytologic study of washings from the maxillary sinus advocated recently by Fitz-Hugh and Moon and Lupton.[9] Robinson[10] points out that aspiration biopsy is helpful when the pathologist is sufficiently experienced to evaluate the results of the cytological technique.

Schall[8] indicates that when cancer is superimposed upon polypoid ethmoiditis it may be suspected if there is heavy bleeding after polypectomy. Tumefaction at the upper and inner part of the orbit, edema of the eyelids, downward and outward displacement of the eye globe and lacrimation; soft, fragile anterior pole of the right middle turbinate and bulla ethmoidalis, heavy bleeding during the operation, and the X-ray picture point in our case toward the ethmoid labyrinth as the primary site of the epidermoid carcinoma which invaded the nasal cavity and the orbit.

PATHOLOGY.

According to Barnhill,[11] "The pathology of these tumors in the nasal accessory sinuses is the same as that of malignant tumors found elsewhere. In carcinoma the infiltrating cells arise either from the surface or glandular epithelium. As infiltration progresses, and frequently the invasion is slow, the adjacent bone and soft structures become involved, causing ulceration and destruction of tissue." If and when the nasal polyp became malignant in our patient is impossible to ascertain. The malignant growth may have the appearance of an innocent nasal polyp which on histopathological examination happens to be epidermoid or other carcinoma. In Evans' (from Ewing[2]) opinion we know little about the relation of nasal polyps to malignant neoplasms.

What is more important, there is no definite limit between nasal polypi which arise from chronic inflammation and malignancy whose etiology is obscure.

It is interesting to note that according to Ringertz[3] 30 per cent of Schneiderian carcinoma, which is a highly anaplastic cancer of the nasal passages, is associated with nasal polypi. Phahler and Vastine[12] state that nasal polyposis is present in

about a third of the cases of squamous carcinoma, but the incidence of nasal polyposis is less frequent with squamous carcinoma than with papillary or cylindric cell carcinoma.

Although nasal polypi are a pseudoneoplasm preceded mostly by chronic sinusitis, they are frequently found as a complication of malignancy like in the instance being reported. The question arises whether in our case a nasal polyp became malignant and invaded the ethmoid sinus and orbit, or whether the mucous membrane of a chronic infected ethmoid labyrinth turned to malignancy, and the nasal polypi became involved in the same process per continuitatem. True malignant transformation of a nasal polyp is exceedingly rare. Heymann (cited from Evans[2]) found only three doubtful cases of such transformation. More probably a chronic polypoid ethmoiditis is the condition which preceded the malignant process in our patient. The continuous irritation and infection of the polypous degenerated mucous membrane of the ethmoid sinus and of the nasal polypi could result in malignancy.

Eggston and Wolff[13] explain such process as follows: The mucous membrane of a chronic infected sinus and the nasal polypi which are the product of chronic sinusitis are covered normally by columnar epithelium. Because of trauma and infection the columnar epithelium may lose its cilia, the surface of the mucous membrane or polypi may become ulcerated, and metaplasia may take place from columnar to stratified squamous type with further change to epidermoid carcinoma. Tumors arising in the ethmoid labyrinth may invade the orbit or vice versa; tumors arising in the orbit may invade the nasal sinuses. In MacComb and Martin's[14] series of cancer of the nasal cavity, 50 per cent of the presented cases extended directly into the orbit, and in 25 per cent the invasion through the ethmoids to the floor of the orbit was demonstrated by Roentgenologic study.

In our patient the floor of the orbit was intact, the malignant process being expanded from the ethmoid labyrinth to the roof and superior nasal quadrant of the orbit in the direction of the frontal sinus.

TREATMENT OF PARANASAL CANCER.

A survey of available literature from 1932 to 1949 concerning treatment is given below. As with cancer elsewhere in the body, treatment depends upon the nature, location and stage of the malignant process, age and general condition of the patient, presence or absence of metastases, and finally upon the attitude of the surgeon.

According to Beck and Guttman[15] our treatment should vary with the histopathologic data. Highly cellular anaplastic tumors should not be operated upon and radium and Roentgen therapy only is indicated. Houser[16] is of the opinion that surgical procedures must be employed only to institute adequate drainage and in order to expose the tumors of the paranasal sinuses to direct irradiation, which is the only proper treatment. Nash[17] outlines the typical management of a malignant disease of the paranasal sinuses. This includes X-ray study with lipiodol, external opening into the sinus for biopsy, drainage and radiation with or without diathermy. It is necessary to consult a pathologist as to the nature of the growth and prognosis. Breeding[18] recommends intranasal, external or palliative surgery in "not hopelessly inoperable" cases to be followed by radium and Roentgen therapy and diathermy. For intractable pain — intracranial division of the Vth or IXth nerves.

MacComb and Martin's[14] scheme of treatment includes general hygienic measures, high voltage Roentgen radiation of the sensitive primary lesion, surgical treatment of the radioresistant original lesion when irradiation alone is inadequate; postoperative high voltage Roentgen radiation; treatment of recurrences, and treatment of metastases. Watson[1] advocates surgical removal of a small localized cancer of a paranasal sinus if it is discovered early and if there is a reasonable assurance of further tumor control. In moderately advanced cases either radiation by interstitial radon, followed by cautery excision of the tumor and involved bone, is advisable; or external Roentgen treatment with surgery, restricted for the establishment of drainage or for control of hemorrhage, is indicated. In desperately advanced cancer of the paranasal

sinuses only drainage conservatively established is necessary. Schall[8] believes that adequate drainage should be established independently of which type of treatment is employed because the patient with proper drainage suffers less during radiation. In an anaplastic tumor he advocates irradiation (external or interstitial). If the tumor does not respond to radiation, surgery with irradiation is advisable.

If the orbit is seemingly involved, as it is in the case reported herein, or even when it is questionable whether or not the orbit has been invaded, Schall advocates the exenteration of the orbital contents, removal of the floor of the orbit to explore the antrum, and removal of the lateral orbital wall to exenterate the ethmoidal labyrinth and create a better access to the frontal sinus if necessary. Where there is involvement of the ethmoidal and maxillary sinuses without extension into the orbit any radical sinus operation is indicated with electrocoagulation of the entire operative cavity and insertion of radium to prevent dissemination of the growth and metastases.

In cases of malignant tumors of the nasal cavity in which there is no Roentgenologic evidence of bone invasion Havens and Thornell[19] advise a frontoethmoidal approach. They believe, however, that the determination of the degree of malignancy is a deciding factor in selecting the treatment of choice. Robinson[10] advocates radical surgery and radiation where the tumor is not too extensive and is not anaplastic. For anaplastic rapidly growing tumors Roentgen therapy should precede operation. Such preoperative treatment causes regression of the tumor, which may be more extensively removed during the forthcoming operation. Intracavitary radium treatment should follow operation. Preoperative palliative Roentgen therapy is indicated in the advanced cases. Chemotherapy and frequent irrigations are invaluable in controlling secondary infection and bone necrosis.

Abdeen[20] feels that the choice of treatment is of lesser importance since surgical excision and radiation used alone or in a combination give in most instances the same final bad results due to the reappearance of the primary visible and secondary invisible lesion.

Schall and Brewer[21] have found in cases with superimposed acute infection the following treatment to be effective: Roentgen radiation supplemented when necessary by surgery for drainage, and large doses of antibiotics and chemotherapy. The treatment of the recurrence of the primary tumor is only palliative. Finally, Capps[7] believes that "pure surgery is now a thing of the past." Nowadays diathermic cutting and coagulation surpass surgery. If the orbit has been invaded, it should be exenterated and plastic surgery employed. Best results are obtained by a combination of radiation and electrosurgery. The same author advocates irradiation with deep X-ray, followed by diathermy exenteration and if necessary by intracavitary radium treatment. Metastases to the lymph glands of the neck should be treated by irradiation rather than by bloc dissection.

A course of deep X-ray treatment directed to the right paranasal sinuses and nasal cavity was performed on our patient in Saskatoon Cancer Clinic. According to Dr. C. Burkell two fields were used, 10 x 6 cm. each; one was directed from the right face anteriorly and the other from the right face laterally. The tumor was estimated to be 8 cm. deep from the face of each of these fields. A total dose of 5,000 Roentgens was applied in this three-week course of treatment. The maximum skin dose was 5,100 Roentgens. The skin reaction was moderate with moist desquamation. Three and one-half months after completion of the treatment the skin of the treated area was well healed with tanning. The patient had no trouble with his right eye except tearing and loss of lashes; his right nasal passage being clear with no discomfort. The regional lymph nodes were not enlarged and the general condition of the patient was satisfactory (Dr. A. G. Genereux).

COMMENT.

In conclusion I believe that in the case presented the primary site of origin of the epidermoid carcinoma was the right ethmoid labyrinth and that the malignant disease was complicated by nasal polypi rather than a nasal polyp turned to malignancy. We could also presume that this epidermoid car-

cinoma of the ethmoid labyrinth was associated with ethmoidal sinusitis, polypoid in nature. In making a proper diagnosis the radiograms were suggestive only and not conclusive, since in a Roentgenogram the neoplastic and nonneoplastic masses cannot be differentiated. The only suggestive sign of malignant diseases in the Roentgenogram was erosion of bone. Extensive erosion of a large portion of the superior nasal quadrant of the right orbital margin and portion of the orbit indicated invasion of the orbit; but it did not necessarily mean that the process was neoplastic. The symptoms of early malignant disease in this site did not differ probably from those of chronic polypoid ethmoidal sinusitis, and this is the reason why here an earlier diagnosis of malignancy could not be hoped for.

Surgical exploration was attempted in view of the destruction of the orbit seen in the Roentgenogram. Profuse bleeding during the operation, fragility of the bulla ethmoidalis, and suspicious macroscopic appearance of one of the removed polypi directed the diagnosis toward malignancy; but the true nature of the disease was disclosed only by biopsy.

The survey of the current trends in treatment of sinus cancer brings to the conclusion that for our patient with highly malignant tumor and extensive destruction of the adjacent bony structures the only appropriate treatment is high voltage deep X-ray radiation.

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SQUAMOUS CELL CARCINOMA OF THE TYMPANIC MEMBRANE.*

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Opinions differ on the frequency of cancer of the auricle and of the external auditory canal. Some workers in this field of research (Peele and Hauser, 1941; Sylven and Hamberger, 1950) claim such invasive growths to be fairly rare, but according to others (Driver and Cole, 1942) and to the Ninth Annual Report of the National Radium Commission, London (1937-1938) about 3 to 5 per cent of all malignant lesions of the skin are seen in the outer ear and in the external canal.

Although nothing definite is known of the etiology of cancer of the outer ear, prolonged irritation from chronic suppuration is widely believed to be a contributory cause (Towson and Shofstall, 1950). Usually the lesion is not recognized until it has involved both the external canal and the middle ear. This accounts for the lack of knowledge of the point of origin of such tumors and for the apparent absence of reports of primary cancer of the drum. Eggston and Wolf (1947) described two cases of malignant tumor of the tympanic membrane, but both were secondary growths. In one a basal cell cancer had grown from the external canal through the drum to the middle ear. In the other case a fibromyxoma had spread from the middle ear through the drum to the external canal.

As the literature contains no description of cancer localized solely to the tympanic membrane, the following case report was thought to be warranted:

*From the Oto-Rhino-Laryngologic Clinic of the University of Lund, Sweden.

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CASE REPORT.

A 70-year-old man, who had a morbid fear of cancer, was examined at the E.N.T. Department on several occasions. Nothing remarkable was observed until 1948, when left otitis externa was seen. The inflammation responded favorably to the instillation of alcohol and boric acid, but persistent itching was claimed. Although he was instructed not to pick or scratch the ear, his mental makeup was such as to suggest that he probably did so despite his statement to the contrary. In September, 1950, suppurating otitis externa recurred, and itching became more intense. At that time both the upper quadrants were reddened as in myringitis. Boric acid was again instilled, the discharge practically cleared up, but the changed appearance of the drum persisted. A month later, suppuration again recurred, granulation progressed and obstructive deafness was noted. Granulation gradually advanced, and in December a biopsy was done.

Pathologist's Report (Ahlström): The excised fragments of the drum are built up of irregular squamous epithelial formations consisting of slightly polymorphous, spinocellular elements with chromatin-rich nuclei

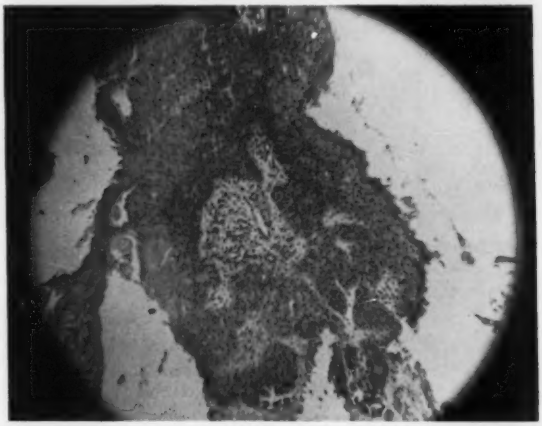

Fig. 1a. Microphotograph of a section of the first biopsy specimen removed from the tympanic membrane.

and scattered mitoses. The epithelium shows a marked keratinizing tendency with the formation of beads. In some areas the line of demarcation between the epithelium and the surrounding tissue seems to be ill-defined and suggestive of infiltrative growth. The stroma is richly vascularized and contains a dense accumulation of leucocytes. The picture does not permit a definite exclusion of epithelial atypia due to chronic inflammation, but the number of mitoses, the cellular atypia and the tendency to infiltrative growth argue strongly for malignancy.

As malignancy was not certain, expectant treatment was given. The granulation, which was strictly confined to the drum, began to grow quickly, and after 14 days biopsy was again done.

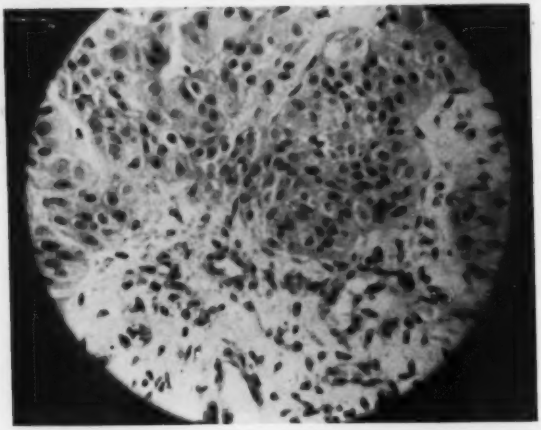

Fig. 1b. Microphotograph of the same section as that shown in Fig. 1a, but under higher magnification.

Pathologist's Report (Mellgren): The fragments of the drum show practically the same picture as before; but now the epithelial growth is richer, the atypia is more pronounced, the keratinization is more advanced, the mitoses are numerous, and the infiltrative growth is more distinct. There is no longer reason to doubt the diagnosis.

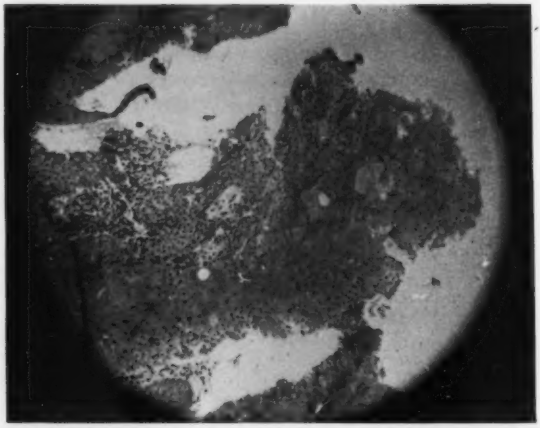

Fig. 2a. Microphotograph of a section of the second biopsy specimen removed from the tympanic membrane.

Diagnosis: Squamous cell carcinoma.

As the tumor had involved only the drum, preoperative radiation was not given because of the risk of radiation necrosis of the auricle and of the cartilage of the external auditory canal; therefore, it was decided to operate. The inner third of the external canal was removed together with the drum and the surrounding bone *en masse*. Postoperatively the

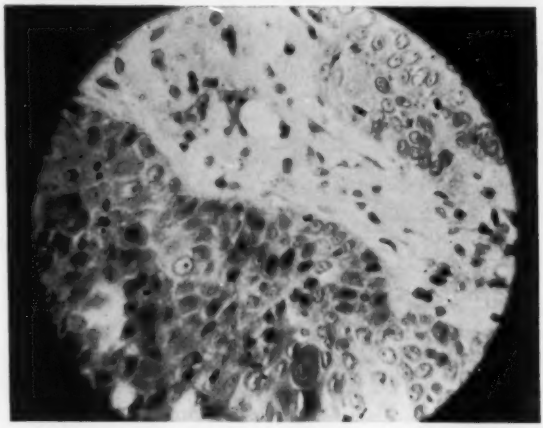

Fig. 2b. Microphotograph of the same section as that shown in Fig. 2a, but under higher magnification.

patient received radiation therapy. Macroscopically, the growth had not encroached upon neighboring parts: this was confirmed at microscopic examination, which also showed that it was localized to the external layer of the upper half of the drum.

In this case the lesion may probably be ascribed to continual traumatization of the drum from irritating picking of the ear. The drum and the adjacent portion of the external canal were removed *en masse*, and the patient received radiation therapy postoperatively. Examination one year after operation revealed a healed cavity with no signs of recurrence of the tumor.

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THE REACTION OF TULLIO AND THE FENESTRATION OPERATION.

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INTRODUCTION.

If an aperture is made in a pigeon's osseous semicircular canal, the sound-waves give a distinct vestibular reaction. This reaction is effected by the fact that the crista of the fenestrated semicircular canal is stimulated by the sound. This reaction was first described by Tullio.[1] Making an aperture in a semicircular canal has become a very frequent operation as a treatment of clinical otosclerosis. The question arises why, as a rule, with this fenestration, these vestibular reactions to sound cause so little or no trouble at all. This question will be answered first in paragraph 1; in paragraph 2 some new particulars concerning Tullio's reaction, which are of interest for the theory of the inner ear function, will be discussed.

1. Tullio gave an excellent description of the reactions after the fenestration of the various semicircular canals of pigeons. The head-deviation and the nystagmus always have their course in the plane of the operated semicircular canal. They are typical reactions of the crista and they continue to exist after removal of the cochlea. A reaction of the utriculus can also be evoked by making an aperture in the vestibulum or, when an aperture in the semicircular canal exists, by eliminating the function of this crista by means of a careful section of the semicircular canal,[2] or, in the posterior semicircular canal by extirpation of the membranous ampulla.[3] These experiments were repeated by only a few investigators: Jellinek,[4] Dohlman,[5] Huizinga.[6]

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Tullio also ascribed a great acoustic importance to these reactions; according to his findings they are, among other things, important for the determination of the direction of the sound. He thought that fenestration increased the irritability of the crista concerned, thus considerably intensifying normal reaction. This explanation is certainly not correct. Tullio's reaction is brought about by the fact that the aperture in the semicircular canal exists, now the sound vibration causes movement of the inner ear fluid in the vestibular labyrinth. Under these abnormal anatomical conditions the crista is stimulated by sound just as the cochlea is, under normal conditions, because of the round window. Tullio's reaction is proof of the importance of the presence of a second window. For stimulation of the sensulae of the inner ear in general by sound, a movement of the liquor labyrinthi is absolutely necessary. This very important function of the round window under normal conditions is sometimes doubted.

When performing experiments on the vestibular organ of pigeons, Tullio's reaction is a very good indicator for ascertaining whether the function of the crista concerned is present. As has already been remarked, the head-movements of the pigeon are very characteristic for the various semicircular canals. Tullio's reaction can also be used for quite different experiments, *viz.*, to inform us of the importance of the middle ear apparatus. The effect of disturbing the normal sound transmission to the oval window via the drum and columella (the only middle ear bone in birds), can be determined. These experiments were performed with van Eunen and Huizing.[7] In the first place, they necessitated the determination of the threshold of these reactions.

As the minimum response of the cochlea is determined for the various frequencies and is plotted as an audiogram, so a similar procedure is also possible for Tullio's reaction. Obvious reactions were obtained at frequencies of 100 to 3,200 d.v.; at a higher level the reaction becomes very weak. The minimum reaction was determined in this way: the fenestrated pigeon was put in front of a loudspeaker through which various frequencies were presented to the ear. At

exactly the same distance stood the microphone of a sound level meter. To the head of the pigeon a pointer was fastened; a slight deviation due to Tullio's reaction could thus be easily observed. In this way a threshold curve could be made. It appears that when the thresholds in decibels obtained with frequencies of 100 to 3,200 d.v. are plotted graphically, a curve of a characteristic form is always obtained. The curves of Fig. 1 of the three different canals are clear

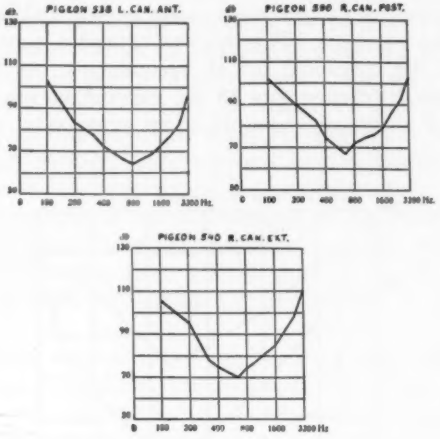

Fig. 1.

examples of it. The minimum value of the reaction with 100 d.v. nearly always lies in the region of 100 db and decreases rapidly with the rise of the frequency, to reach at 600 to 800 d.v., the lowest point at about 70 db. A prompt increase of the threshold value follows and this in such a manner that the last determination, *i.e.*, at 3,200 d.v., usually lies higher than the original point. The curve is the same for the different semicircular canals. Fig. 1 shows the threshold curve for the anterior, posterior and external canals, respectively. The optimum of the reaction always lies at the same point, *viz.*, 600 to 800 d.v.

Tullio's reaction was quantitatively determined in 92 pigeons. In 25 of these experiments extremely high values were found, the minimum lying at 80 db or higher. This could always be explained by the fact that blood had entered the middle ear, thus disturbing the sound transmission, or that the aperture in the semicircular canal had turned out too large so that the function of the crista had suffered by loss of much perilymph. For the other 67 pigeons the average lay at 69 db, spread between 61 and 79 db.

In 20 pigeons, curves were made before and after extirpation of the eardrum. This always caused a considerable elevation of the threshold for all frequencies. At the optimum its average difference was 28 db, varying between 20 and 40 db. Fig. 2 shows an example of an experiment of this kind.

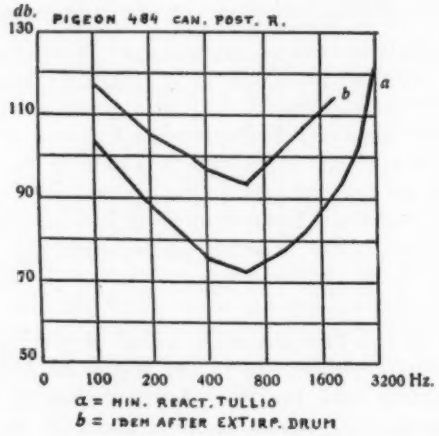

Fig. 2.

In this case a fistula had been made in the posterior canal of the right side. Graph A is the minimum reaction for the various frequencies before the removal of the eardrum; Graph B was recorded after its removal. At the optimum the difference amounts to over 20 db. This experiment once more

shows the great importance of the eardrum for the transmission of sound to the inner ear. Obvious individual differences exist. Perhaps these also exist in man, thus explaining, beside other possibilities, the very divergent effects of a total defect of the eardrum on the hearing in man.

We see, therefore, that when one of the semicircular canals has been fenestrated in the pigeon, under ideal anatomical circumstances (no infection, no labyrinth degeneration), it shows no vestibular phenomena with a sound of less than 70 db and after removal of the eardrum of less than nearly 100 db. Are these values of any significance for man? The anatomy of the middle and inner ear of the pigeon shows some differences from that of the mammals and one might think that it is impossible to make comparisons. It is very important that Wever, Laurence and Smith[8] showed that in the cat removal of the eardrum and ossicular chain caused a rise in the threshold of electric response of about 28 db. The values of the pigeon and the cat, therefore, are exactly the same. Because of this fact, a much greater significance can be attached to these experiments on the pigeon.

In the pigeon, section of the handle of the columella has the same effect as removal of the eardrum. In either case, the conduction apparatus to the oval window is disrupted. Probably the round window under these circumstances becomes the way of entrance for the sound. In all probability such is also the case after fenestration in man.

A complete restoration of the auditory function by means of fenestration is, however, impossible, as the ossicular chain, which, together with the eardrum, serves as a mechanical transformer and also as an amplifier, is interrupted. The animal experiments are in complete concordance with the dissertations and findings of Walsh and Davis[9] concerning the limits of improvement of the fenestration. The mean postoperative hearing loss for 69 cases of pure conductive deafness was 26.4 db. A greater hearing loss must be ascribed to nerve impairment or surgical "accidents." This result of 26.4

db, therefore, completely agrees with our expectations on the grounds of theoretical considerations and from the animal experiments both on the cat and the pigeon.

If we assume the relations in man to be the same as in the pigeon, then, according to these data, a fenestrated patient with an ideal history would show Tullio's reaction only at an intensity of nearly 100 db and for a few frequencies only. This explains why patients after fenestration are not troubled by vertigo due to sound. In reality the threshold lies still higher than the already considerable intensity of 95 to 100 db.

Jongkees and Hulk[10] stated that after the fenestration operation, a clearly diminished irritability of the vestibular organ is found after rotation in the horizontal plane; this is also true of the crista externa. As a matter of fact, this was to be expected. Most patients are troubled by vertigo after the fenestration and show a spontaneous nystagmus that must be explained by loss of function of the crista externa. This is especially so when the fenestra includes part of the ampulla, as is generally done with the new operative technique according to Lempert. This strongly diminished irritability can also be shown in the pigeon by fenestration of a semicircular canal and an ampulla. When this is done a much higher threshold for the rotatory reaction is found immediately after the operation. Complete recovery does not always follow even after a lapse of some time.

There is another point of still greater practical importance. The threshold of value of 70 db in the pigeon was found immediately after making the aperture in the semicircular canal. The next day the reaction considerably diminishes and then gradually disappears. In the fenestration operation on man the aperture is closed with a skin flap. Owing to this, the threshold will probably also be much higher.

From all this it is clear why little or no vertigo is experienced with loud sound after fenestration. This is fortunate, for, were the relations different, fenestration would be impossible owing to the considerable vertigo from sound. We found patients with very good results after fenestration, in

whom at an intensity of 115 db Tullio's reaction could not be produced. The fistula test always gave clearly positive results. Apparently the mobility of the new window was sufficient for stimulation of the cochlea by sound, but not for stimulation of the crista. At still higher intensities (in practice patients are not subjected to these) it must be expected that Tullio's reaction does become positive. On the other hand, Benjamins[11] formerly described a patient from this clinic, having a fistula in the horizontal semicircular canal due to cholesteatoma but having fair hearing (whispered speech, low, 1.5 m.; high, 3 m.), in whom at an intensity of 110 db Tullio's reaction could be clearly produced. After the radical operation the hearing was considerably diminished, so the sound transmission to the inner ear had been seriously damaged; Tullio's reaction was negative, whereas the fistula test gave positive results.

2. Investigations by Bleeker and de Vries[12] from this laboratory demonstrated that in the pigeon microphonic effects can be produced by sound from the cristae as well as the cochlea. For the various frequencies up to c. 2,200 d.v. a sine-wave can be produced from the crista of the fenestrated semicircular canal of the same frequency as that of the stimulating tone and differing little in size from the cochlea effect. These experiments were confirmed and largely extended by van Eyck.[13] Experiments by Vrolijk and de Vries[14] demonstrated that an effect can be produced from the utriculus. They could also produce a microphonic effect from the isolated crista in pigeons. At an earlier date[3] it was shown that it is possible to remove the crista posterior in pigeons, without the remaining part of the pars superior losing its function. The opposite was also possible in a number of cases: the two anterior ampullae and the utriculus were removed, thus leaving an isolated crista posterior. In a series of 30 pigeons it was possible eight times to deduce a very clear microphonic effect and a weak one six times. It is not surprising that in a great number of cases after such an extensive operation (flowing away of the perilymph and endolymph) the function of the crista posterior is also damaged. The existence of a crista-effect, therefore, cannot possibly be doubted any more.

A strong parallelism exists between the crista-effect and Tullio's reaction: if the former is strong, then the latter is so also; if Tullio's reaction is completely absent, a crista-effect cannot be produced either. The threshold of Tullio's reaction lies much higher, however, than that of the crista-effect. Tullio's reaction begins at the optimum at c. 70 db, clear potential fluctuations usually can be deduced at as low as 35 db. The sensitiveness of the apparatus in use plays an important part. It clearly shows that a certain connection exists between Tullio's reaction and the microphonic crista-effect by comparing

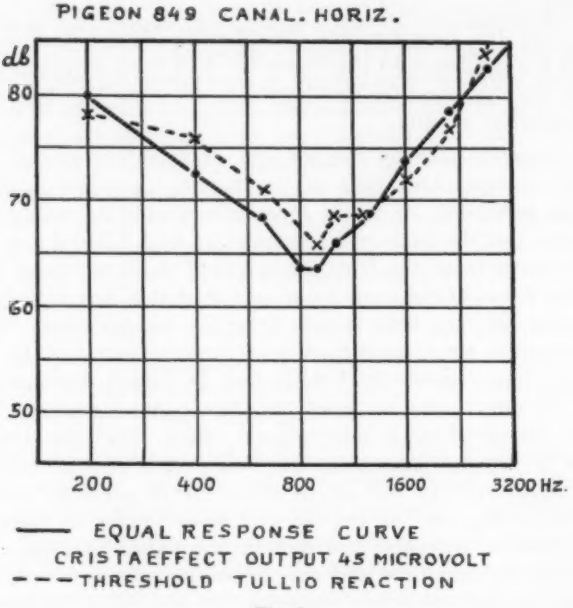

Fig. 3.

the threshold curve for the various frequencies to the equal response curve of the latter. The equal response curve of the crista-effect was already described by Bleeker and in this connection was more extensively checked by Vrolijk. In Fig. 3, both curves of a certain pigeon are plotted next to each

other. The continuous line is the equal response curve for an output of 45 microvolt. The dotted line is the threshold of Tullio's reaction. The parallelism is obvious without comment.

Just as with the cochlea-effect, it is still completely uncertain with the crista-effect if any physiological meaning may be ascribed to this fluctuation in potential. It is quite possible that in this case we also encounter an attendant phenomenon; but just as the cochlea-effect, it certainly has its great scientific importance. At any rate, the existence of this effect proves that in Tullio's reaction the crista vibrates synchronously with the frequency presented to the ear. Certainly no adequate stimulation of the crista exists, owing to the origination of endolymph currents, according to Tullio. Formerly, somewhat under the influence of Wittmaack's views, I thought that Tullio's reaction was caused by pressure. Particularly if one assumes that the influence of sound on the inner ear causes eddies, one can imagine that under these circumstances the crista is subject to pressure. Now that it has been proved that the crista actually vibrates, the following explanation proposed by Dohlman is much more plausible. The crista quickly moves to and fro. One might imagine that these quick movements in both directions would neutralize each other, but this is not the case, however. The movement to one side has a far greater effect than that to the other side. Cumulation causes the strong reactions, that, owing to the influence of sound are also possible on the crista. With the horizontal crista the ampullopetal movement gives the stronger reaction. With the vertical semicircular canals this is just the reverse, the ampullofugal movement being more active. This is Ewald's second law, which was actually demonstrated on the pigeon. Indeed, van Eyck and also Vrolijk were able to confirm Ewald's second law for pigeons by means of electrophysiological investigations.

As was described above, Dohlman's explanation of the mechanism effecting Tullio's reaction at present seems to be the most correct one. This also gives a strong indication of the validity of Ewald's second law. If we study the head-

movements of the pigeon in Tullio's reaction it appears that, the horizontal crista being stimulated, the head moves in the horizontal plane to the opposite side (the nystagmus is homolateral). When the anterior crista is stimulated, the head moves backward; when the posterior crista is stimulated, it moves forward. This is completely in accordance with the view that in the horizontal semicircular canal the ampullopetal movements gives the stronger effect and in the vertical semicircular canals, the ampullofugal one.

Of late, doubts were frequently expressed as to the validity of Ewald's second law. It is difficult to prove conclusively whether or not it exists in man, but with the pigeon this can hardly be doubted. The corresponding results in comparative labyrinth-physiology tell in favor of the assumption that Ewald's second law also applies to man.

SUMMARY.

Tullio's reaction is discussed. If, in test animals, the pigeon in particular, a fistula is made in the osseous semicircular canal, a very strong vestibular reaction is caused by sound, owing to stimulation of the crista. The explanation of the effect of this reaction must be quite different from the one suggested by Tullio. The reaction has great scientific value. With pigeons it can be used as an indicator of the effect of various operations on the conductive apparatus. In this way it could be shown that removal of the eardrum or section of the columella means a loss of 28 db. The remarkable fact is discussed that after the fenestration operation patients are little troubled by vestibular reactions, even with loud sounds. A few new points of view concerning Tullio's reaction, cleared up by the microphonic effect of the crista, are discussed. Ewald's second law is certainly valid for pigeons.

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LARYNGECTOMY TUBE FOR OXYGEN THERAPY.*†

ALBERT H. ANDREWS, JR., M.D.,
Chicago, Ill.

The addition of oxygen to inspired air following major surgical procedure is of recognized value in speeding recovery and preventing complications. A special inner tube has been designed for this purpose, which has an extension with a side arm and cap.‡ The straight part of the extension is attached by rubber tubing to the tracheotomy type of meter mask, and the mask is pinned to the patient's gown to prevent inadvertent kinking of the tube. No appreciable respiratory obstruction is produced. The side arm of the tube is capped except when aspirating the trachea, thus permitting aspiration without disturbing the oxygen apparatus. The tube locks in place by conventional rotating collar and may be removed easily for cleaning (see Fig. 1).

During the first hour following surgery, the meter is set for "100" per cent oxygen. Water is placed in the meter so as to produce a relative humidity of about 40 per cent. After the first hour, the meter is adjusted for 50 per cent oxygen. A heat type humidifier with long delivery tube is adjusted so that the steam is delivered close to the air intake of the meter. This produces warming and high humidification of the gas. This is continued for 12 hours, and then the special inner tube is replaced by the regular one. No harmful effects or drying

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†From St. Luke's Hospital, Chicago, Ill.

‡Produced by the George P. Pilling & Son Co., Philadelphia, Pa.

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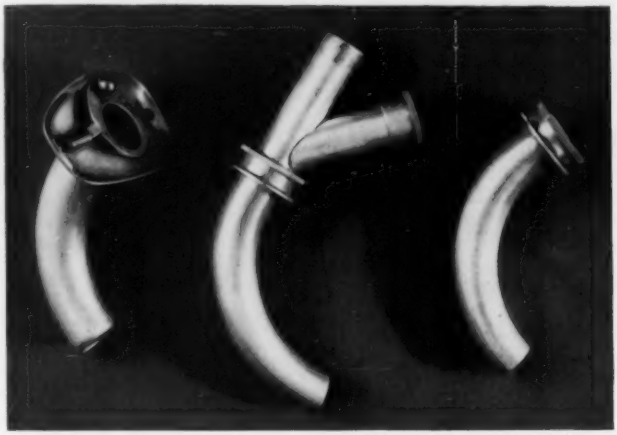
of secretions have been observed. It is thought that wound healing and the general condition of the patient have been improved by this procedure.



(A) (B) (C)

Fig. 1. Laryngectomy tube for oxygen therapy. (A) Standard Lewis No. 8 laryngectomy outer tube. (B) Special inner tube to which the meter mask for tracheotomy is attached. Side arm is used for catheter aspiration with the cap removed. (C) Regular inner tube.

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Tonamic Model 50.
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Tonemaster Model Royal; Model Cameo.
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